Ontario

ROYAL COMMISSION OF INQUIRY INTO CERTAIN DEATHS AT THE HOSPITAL FOR SICK CHILDREN AND RELATED MATTERS.

Hearing held
21st floor
180 Dundas Street West
Toronto, Ontario

The Honourable Mr. Justice S.G.M. Grange	Commissioner
P.S.A. Lamek, Q.C.	Counsel
E.A. Cronk	Associate Counsel
Thomas Millar	Administrator

Transcript of evidence
for

June 11, 1984

VOLUME 152

OFFICIAL COURT REPORTERS

Angus, Stonehouse & Co. Ltd.,
14 Carlton Street, 7th Floor,
Toronto, Ontario M5B 1J2

595-1065

ANGUS, STONEHOUSE & CO. LTD.
TORONTO, ONTARIO

ROYAL COMMISSION OF INQUIRY INTO CERTAIN DEATHS AT THE HOSPITAL FOR SICK CHILDREN AND RELATED MATTERS

Hearing held on the 21st Floor, 180 Dundas Street West, Toronto, Ontario, on Monday, the 11th day of June, 1984.

THE HONOURABLE MR. JUSTICE S.G.M. GRANGE - Commissioner

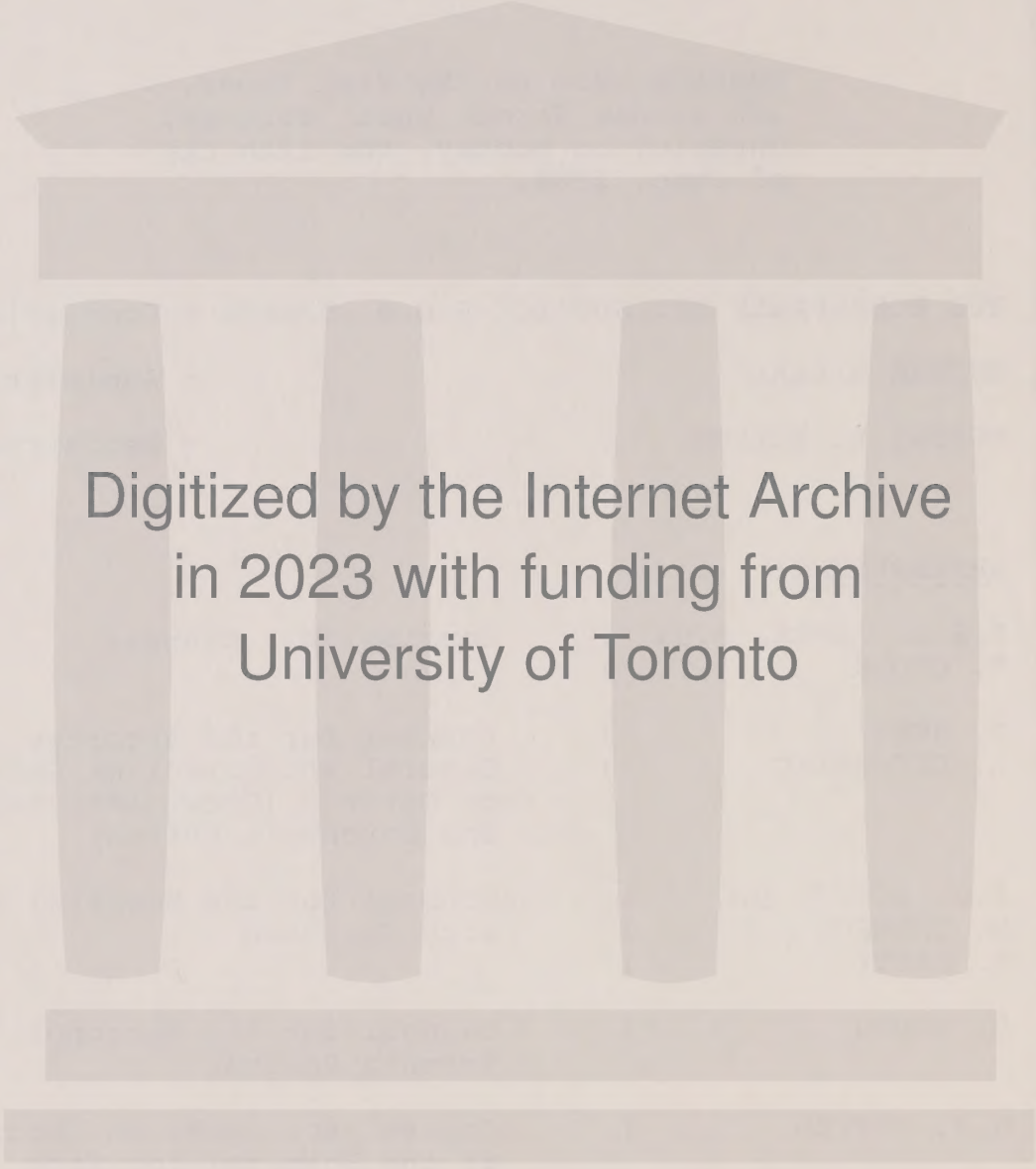
THOMAS MILLAR - Administrator

MURRAY R. ELLIOT - Registrar

APPEARANCES:

P.S.A. LAMEK, Q.C. E. CRONK	Commission Counsel
D. HUNT L. CECCHETTO	Counsel for the Attorney General and Solicitor General of Ontario (Crown Attorneys and Coroner's Office)
I.G. SCOTT, Q.C. M. THOMSON R. BATTY	Counsel for the Hospital for Sick Children
D. YOUNG	Counsel for the Metropolitan Toronto Police
W.N. ORTVED	Counsel for numerous Doctors at the Hospital for Sick Children
D. BROWN	Counsel for Susan Nelles - Nurse

(Cont'd)



TRANSCRIPTION BY MEMBER
ANGUS, STONEHOUSE & CO. LTD.
TORONTO, ONTARIO

APPEARANCES: (Cont'd)

G.R. STRATHY E. FORSTER	)	Counsel for Phyllis Trayner - Nurse
J. OLAH	)	Counsel for Janet Brownless - Nurse
S. LABOW	)	Counsel for Mr. & Mrs. Gosselin, Mr. & Mrs. Gionas, Mr. & Mrs. Inwood, Mr. & Mrs. Turner, Mr. & Mrs. Murphy (parents of deceased children)
W.W. TOBIAS	)	Counsel for Mr. & Mrs. Hines (parents of deceased child Jordan Hines)

VOLUME 152

I N D E X

A
D/cr

THE COMMISSIONER: Yes, Mr. Lamek.

ARGUMENT BY MR. LAMEK (Continued)

MR. LAMEK: Mr. Commissioner, when we came to an untimely end on Thursday I just finished dealing with the four children in respect of whose deaths Miss Nelles was charged and I told you that I proposed to move to three children, in whose bodies digoxin was found, although it had never been prescribed.

The first of those whom I start this morning is Jordan Hines.

The nursing note of Nurse Reaper for the long night shift of March 7th to 8th, the night on which Jordan Hines died, is found on page 35 of the chart and discloses that at 4 o'clock that morning Jordan Hines' apex was 182 and regular. That is the second last line in the note, sir.

"At 0400 hours apex 182 and regular. Respiration 54 and no noted distress."

Ten minutes later he suffered a cardiac arrest. 35 minutes after that he was dead.

Hines as a patient on Ward 4B, the Trayner nursing team was on duty that night on Ward 4A. In the early part of the shift, as is apparent

from the rest of Nurse Reaper's note, Baby Hines had a regular heart rate. He fed well, vomited a small amount at 3:00 a.m. He was alert, active, voiding well. His chest was slightly congested she records. He certainly appeared to be at no risk of dying that night.

Dr. Rose, who had seen the baby briefly on rounds on Saturday, and her evidence is found at Volume 37, pages 7301 to 2, thought he was somewhat unstable, but not critical and she did not then think, she said, that Hines was a terminal case.

On the Saturday night, on the early Sunday morning she was called at home and told of Baby Hines' cardiac arrest.

Dr. Kobayashi saw the child more frequently and later in the day on Saturday than Dr. Rose did. He said and his evidence is found at Volume 142, beginning at page 2669 and going to page 2674, that on the Saturday morning the baby appeared to him to be, and I use his words "extremely stable". There was no suggestion or indication that Dr. Kobayashi thought that the baby was in a critical condition or at imminent risk of deterioration. He said that he saw Baby Hines several times during the Saturday. He saw no bradycardia nor apneic spells

and, indeed, no such spell was reported to him by the nurses as having occurred.

At 11 o'clock at night he had no concern about the baby's condition, the baby did not appear to have changed since the morning. In the early morning, between 1 and 2 o'clock Dr. Kobayashi made final rounds before going up to bed and he saw Baby Hines. Baby Hines at that time was on cardiac monitor, which showed normal sinus rhythm. There was no feeding problem and again no apneic or bradycardiac spells.

It was Dr. Kobayashi's impression that clinically Baby Hines looked a great deal better than he had when he first had been admitted. He appeared to be improved and stable. Dr. Kobayashi had no concern about him.

He said in Volume 142, page 2680 that he found the Hines' death distressing and shocking and he regarded it as unexpected and that the cause of death appeared to be a puzzle for everybody.

Dr. Kobayashi's impression, in my submission, was correct, that people were indeed puzzled about the cause of Baby Hines' death.

Dr. Rowe said, and this is in Volume

17, page 2888, that he and his colleagues did not know what the cause of death was when that baby died.

I believe it to be fair to say, sir, that there have only ever been two real candidates for the cause of death of Jordan Hines: one, sudden infant death syndrome or two, digoxin toxicity.

The SIDS explanation is essentially based on Dr. Becker's findings at autopsy of changes indicative of earlier hypoxic episodes or in earlier missed-SIDS incident.

As I understand the theory is this: that infants who survive a SIDS episode, which is hence called a missed-SIDS, infants who survive such an episode frequently succumb to a subsequent SIDS episode. The pathological findings made by Dr. Becker were indicative to him of an earlier missed-SIDS episode and hence, the postulating of SIDS, as the cause of Baby Hines' death, not the episode of which pathological findings were evidence, but an episode of the kind known as a matter of experience and observation in the medical world to recur after there has been a missed-SIDS episode.

On the other hand, the digoxin toxicity explanation is based on findings of digoxin in the fixed and exhumed tissues of Jordan Hines,

digoxin never having been prescribed for him.

In my submission, sir, your conclusion as to how and by what means Jordan Hines died, comes down to a choice between those two explanations, for no other explanation has been advanced. It is plain from the evidence of the pharmacologists that the detection of digoxin in fixed, and particularly in exhumed tissues, can disclose little, if anything, as to how much digoxin was present and in what concentrations at the time of death, or as to when the digoxin was received, although perhaps what I call a not-before-time may be put upon the receipt of the digoxin.

Dr. Kauffman, in Volume 72, pages 5763 to 4 suggests not more than 48 hours before death.

Dr. MacLeod's opinion was for a rather long period of time, as you will remember.

Thus the digoxin findings in Hines only strictly indicate that at some point in time, and if you accept Dr. Kauffman's opinion, likely within 48 hours prior to his death, Baby Hines somehow was given digoxin. If that occurred, clearly it was an unprescribed dose, but there is no way of knowing from that fact alone whether the dose was of a

therapeutic size or of a toxic size, whether it was given in error or deliberately or, indeed, whether it played any part in Jordan Hines' death.

The point of the problem is this: I think, sir, if the sudden infant death syndrome explanation does not satisfactorily account for Jordan Hines death, the only other contender is digoxin toxicity, which is made the more likely by the circumstances surrounding the child's death, in my submission.

First, with respect to the SIDS then. We have heard, and we can probably all repeat it in our sleep and probably all do repeat it in our sleep, that SIDS is a diagnosis of exclusion. That is to say, if I understand it, if there is some other condition that may reasonably explain the death, the diagnosis of SIDS is inappropriate.

Dr. deSa's point in his letter, as filed, explaining his point in this regard says, if I understand that all right, that he is not happy with the SIDS diagnosis in the Hines' case, because the toxicological findings raise another possible cause of death, which cannot be and has not been excluded.

In Volume 71, page 5651 Dr. Kauffman had this to say on the question beginning at line 22.

B-1

Dm/ac

"He" meaning Hines:

" He did have some non-specific findings which were suggestive of Sudden Infant Death Syndrome but I didn't place a lot of credence in that because the rest of his history and physical findings in the Hospital of course really didn't fit that at all, that syndrome at all. "

And he goes on at page 56, 57, to explain what he meant by that and beginning at line 17:

" SIDS, I think is a clinical diagnosis. It is not a pathological diagnosis. It is a clinical diagnosis which may or may not be supported by pathological findings at death.

SIDS by definition is Sudden Infant Death in an infant who appeared to be well up until the time he was suddenly and unexpected found dead for no apparent reason. Frequently there are no specific findings.

" The findings reported in this report have been reported in infants who died unexpectedly and suddenly without any apparent cause and who were perceived to be in perfectly good health at the time they died.

As far as I am concerned if an infant has another illness or symptoms or physical findings which indicate that he is seriously ill, it by definition is not SIDS because there is another cause for the death.

So when this infant came in at 2½ weeks of age with rather profound symptomatology everybody recognized immediately that he was seriously ill. It could be a number of different things but there was no doubt that he was ill and having trouble.

So that along with the findings of abnormal heart rhythm, changes in heart rate and the other symptoms

" that he had, said that something was wrong with him, he is not a baby in normal health and the fact that he died then must be related in some way to his previous illness, and as far as I am concerned cannot be defined as Sudden Infant Death Syndrome.

Q. Dr. Kauffman, to make sure that I understand what you have said, are you saying that the fact of the child's illness on admission to Hospital was sufficient in your view to make a clinical diagnosis of Sudden Infant Death Syndrome inappropriate?

A. No, the fact that he had a pre-existing illness.

Q. Was there any other feature of his clinical condition or any other symptoms which he manifested during life which influenced you in reaching your conclusion in this regard?

A. I couldn't answer that more

" specifically then I have without going through the chart again and refreshing my memory. But in general the answer to your question, it was the fact that although he arrived and was having some arrhythmias, he did appear to be relatively stable and then had a sudden arrest with findings that were compatible with digoxin toxication from which he could not be resuscitated. And then digoxin was unexpectedly found in his tissues when he had never received the drug. And that is a summary of the factors that influenced me. "

Mr. Commissioner, if I understand Dr. Kauffman right, he appears to be challenging the SIDS explanation on two bases. First on the basis that quite apart from digoxin findings Hines was known to have a pre-existing sickness. He was a seriously ill child when he arrived at the Hospital. He appears to be saying that unless it can be demonstrated that that pre-existing illness did not have anything to do with his death, then on that basis alone SIDS is an

inappropriate candidate for cause of death.

He then appears to be saying that quite apart from the pre-existing illness which would make SIDS inappropriate, there are toxological findings of digoxin, together with symptoms of digoxin intoxication, which again unless excluded make SIDS an inappropriate cause of death.

Dr. Hastreiter in Volume 76, pages 6783 to 6784 and elsewhere in his evidence, similarly challenges SIDS as a cause of death. Dr. Mirkin's evidence is to the same effect, and that is found in Volume 87, pages 8916 to 8919.

Frankly, Mr. Commissioner, and it is unfortunate, that the battle lines on the SIDS question appear to pit the physicians at the Hospital for Sick Children against the non-Hospital for Sick Children physicians.

It is my submission that in light of the manner of Jordan Hines' death and nature of his terminal symptoms; the unexpectedness of his death; the lack of any explanation for the death until the SIDS theory was raised; circumstantial matters such as the time of the death and the presence of the same nursing team; the swiftness and irreversibility of the decline of the child; the

presence of digoxin where none was supposed to be, and none ever having been prescribed. In fact if my earlier submission be valid there was on or around the ward someone capable of administering unprescribed digoxin doses for infants; on the basis of all of that, it is my submission that you can and should found that the more probable cause of Jordan Hines' death was digoxin toxicity resulting from the administration of an unprescribed digoxin dose of an unknown size but of sufficient size to cause his death.

While that explanation remains plausible, there is in my submission no room for Sudden Infant Death Syndrome as the cause of death.

If that be acceptable, the remaining question is this, if Baby Hines did then die of digoxin overdose was that digoxin administered by accident or deliberately? One can't be certain of course that Baby Hines was not given another child's digoxin dose by mistake. Although that might perhaps be easier to contemplate, whether or not three other children who also received digoxin when none had been prescribed for them. The possibility that Hines was given digoxin inadvertently in substitution for a drug he was supposed to receive was canvassed with

the pharmacologists.

The only medications prescribed for this baby were ampicillin and gentamicin. Drs. Hastreiter, Spielberg, Mirkin, and Kauffman have all expressed the opinion that errors involving digoxin and either of those antibotics are unlikely. Therefore in my respectful submission you are entitled to find about Jordan Hines that he probably died of digoxin intoxication resulting from the administration of an unprescribed dose and presumably excessive dose of digoxin and that there is no evidence to suggest that that administration was accidental or by mistake.

May I move on to the case of Stephanie Lombardo. This child died, sir, at 4:20 in the morning on December the 23rd, 1980. She was ten days old. She had spent the whole of her short life in the Hospital having been admitted to the Hospital for Sick Children the day she was born. She had severe congenital heart disease and had undergone surgery at the age of four days for a shunt procedure. She did quite well post-operatively and went from the ICU to ward 4A on December the 22nd. There was concern about the size of the shunt and she was on heparin to guard against the shunt becoming

blocked by blood clots.

At page 41 of the chart is the note for the long night shift for the night that she died. It begins at the bottom of the page and it is Nurse Ganassin's note. It records that the baby from 7:00 in the evening until 3:30 in the morning was relatively stable. The heparin was infusing well. The baby was feeding eagerly one and a half to two ounces every three hours. Her apical heart rate was between 144 and 152 throughout that period and regular. Respirations were 50 to 52 a minute, were shallow but she was in no distress and she was pink in room air, became dusky when she was upset. She became restless after the second feed but settled well. In fact being pink in room air, as I understand the medical evidence suggests that there is reasonably good oxygenation of the blood, in other words the shunt is working. At 3:30 in the morning there is a sudden change in that picture. Nurse Ganassin notes that the baby became restless, breathing was very shallow and the apex became irregular and bradycardic.

- - - - -

She was placed on a cardiac monitor, her colour became increasingly dusty, 100 per cent oxygen was given by mask and her extremities became cold and cyanotic. She vomited a small amount of mucus. The doctor was called and a Code 25 was called.

Page 41 is the arrest note. I respectfully draw your attention to the sequence and pace of events. The medical resident notes that he was called at 3:30 about an irregular apex with bradycardia. His observations were that the baby was cyanode, cool extremities, weak pulses, her heart rate was irregular on the monitor, 50 to 180 with variable QRS patterns. I do not know what HS means but it was faint, no murmur heard, which rather suggests that the shunt at that stage was not working. He called a cardiology fellow and a cardiovascular surgeon and tried to get arterial gases and gave oxygen by mask.

At 3:40, 10 minutes after the resident arrived, the baby vomited, was suctioned. At 3:45 she went into cardiac arrest - fibrillation - CPR was started. At 3:48 the arrest team arrived and took over the resuscitation efforts and the parents and Dr. Rowe were informed. Arterial blood gases at 4 o'clock at those levels. The baby was

incubated, defibrillation attempted. From fibrillation she went straight into a complete lack of any heart beat, they were unable to initiate any heart rate and at 4:20 in the morning the resuscitation efforts stopped.

At 3:30 Stephanie Lombardo went into a sudden and terribly steep decline. It was thought at the time that her shunt had closed down and if that were the case her death was entirely consistent with her clinical picture. I suggest, too, that sudden occlusion of the shunt had led to her death could not have been confirmed or rejected unhappily because her parents would not consent to an autopsy.

Fairly there was no reason for the position of the Hospital to doubt that explanation of the death. It had been known that the shunt was not large. The fact that the child was receiving heparin is indicative of the physician's recognition of the danger of the shunt occluding. There was a note in the chart, the resident's note, that when he was called to see the child at 3:30 in the morning no murmur was heard. It appeared that a satisfactory explanation had been provided for her death.

Fourteen months later her body was exhumed and an autopsy was performed on the body. Unhappily we do not know whether it was at that stage

possible to determine the status of the shunt or whether that investigation was ever made. Tissue samples were taken for digoxin. Digoxin had never been prescribed for Stephanie Lombardo and her exhumed tissues, heart, liver, lungs, stomach, showed high concentrations of digoxin as set out in Exhibit 95, and I will summarize them for you, sir. These are all measurements of digoxin, that is RIA and HPLC: in the centre of the heart, 677 nanograms per gram; in the left ventricle, 487 nanograms per gram; in the liver, 354 nanograms per gram; in the lung, 289 nanograms per gram and the stomach contents sample, in the average of 629 nanograms per gram.

Mr. Commissioner, I do not cite those numbers to give them credibility as absolute numbers. Clearly they have none as absolute numbers in any way indicating the concentration at the time of death. But at least one of the pharmacologists and one of the cardiologists have been prepared to pay some attention to the order of those numbers as I shall say in a moment.

With the results of the digoxin assays of the tissuesof Baby Lombardo clearly an entirely new and very different cause of death became possible. The baby should have had no digoxin

in her body. In fact she had digoxin at levels which might suggest that she had had a substantial amount at the time of her death.

Just as all physicians had agreed that Stephanie Lombardo's death was consistent with her clinical condition and with the hypothesis that her shunt had occluded so now they all had to agree that her death, her manner of dying and her terminal symptoms were also consistent with digoxin intoxication. Clearly the child had received digoxin during her life. Equally clearly she was not supposed to have received it. But the same problems of interpretation seem to apply here as they did in the Hines' case. The difficulty is to know when the child received digoxin, how much digoxin she received and the circumstances of her receiving it.

As to the amount of digoxin that she received, Dr. Hastreiter in Volume 76 at page 6787 said this in the course of his direct examination. The numbers reported by Mr. Cimbura's lab had been recited to him. The question at the top of page 6787:

> "Now, you gave evidence at the preliminary inquiry, Dr. Hastreiter, as to the significance of those findings, and that evidence is found

"in Volume 34 at page 16; I won't take the time to read that but I hope I summarize it correctly. It was your view that if the chest fluid in which concentration of 225 nanograms per millilitre was measured, if the chest fluid were blood it would certainly indicate in your mind digoxin poisoning. You didn't know what that fluid really was and it could be contaminated by stomach contents you said, you didn't know what it could be?

A. Right.

Q. You then referred to the recorded tissue concentrations which clearly was also very high and which at that had they been measured in fresh tissue would be strongly suggestive of poisoning.

On the other hand, you recognized in your evidence that dehydration of dessication of the tissues may have served to elevate the digoxin concentrations, but you were still inclined to regard the digoxin levels

"in the exhumed tissues as pointing to the probability that the child had received an overdose of digoxin, but you couldn't, you said, arrive at that conclusion with any absolute certainty?

A. Correct.

Q. Have I reasonably summarized what you recall your evidence to have been?

A. Right.

Q. Are you still of those same views with respect to the toxicological data in the case of Stephanie Lombardo?

A. Yes. I had some reservations about the source, the quality of that fluid.

Q. Yes.

A. And what it was, whether it was contaminated or not. I was concerned about our lack of experience with exhumed tissues, and the fact that possibly dehyrdration or drying of the tissues could have concentrated digoxin and made it appear higher than it actually was. However, it would be

"very, very difficult to explain, even if his levels were within a therapeutic range, you know, why would they be there when this child was not prescribed digoxin. Not only that, but if the child had accidentally received one dose of digoxin you would not expect to have even therapeutic levels in the tissues, and certainly not in fluid if it were blood, and of course we don't know exactly about that. This was the source of my reservation, otherwise there would be very little doubt that there would be 'probable murder'.

Q. But I take it the one clear thing about the findings of digoxin by HPLC and RIA in the tissues of this child is that digoxin was found at all, that is the truly significant thing about the findings is it not?

A. I think more than that, perhaps the fact that these levels are really quite high, you know. I think all levels are considerably above what one would consider normal therapeutic range in these tissues."

" Of course, as I say we don't have a lot of experience with exhumed bodies and it is difficult to interpret these values.

Q. Is there any question in your mind, however, Dr. Hastreiter, but that this child received one or more unprescribed doses of digoxin?

A. No question at all.

Q. And you have to some extent on the basis of the levels recorded here, I suggest you have expressed a very qualified opinion on the quantitative significance of those findings, have you not?

A. Yes.

Q. Not within any range of precision but you have said they are very high. They are higher than you would expect to find in the case of one mistaken dose having been delivered to the child?

A. Definitely.

Q. And I take it they gave you cause for concern as to the likelihood

" of digoxin having been involved in this child's death?

A. Yes. "

Then turning over to page 6791, Mr. Commissioner, at line 3:

" MR. LAMEK: Q. You have said Dr. Hastreiter that the levels recorded in these exhumed tissues were greater than you would expect to see had this child received by error the normal maintenance dose -- I assume maintenance dose of digoxin. Was that your meaning?

A. Right.

Q. Now that rather implies as my learned friend Mr. Scott says that you might have had some expectation as to the levels you would find had the child received a normal maintenance dose in error. Can you give us an idea what you might have expected to find had the child received a normal maintenance dose by error?

A. I think first of all one should

" understand that the ordinary child that is treated with digoxin is not treated with one maintenance dose. The child is usually loaded with the medication first, so-called digitalization, and then maintained on the drug.

If you were to give a child let's say you take any baby and give the baby one maintenance dose of the drug without any previous loading, your blood level would be very low; your tissue levels would be extremely low, and there is no way that you would expect even if you had severe dehydration or crying of these tissues ... "

That may be drying of these tissues.

" ... to have such a high concentration in the tissue. So I can't tell you the exact figures.

I would expect if this was a fresh body, fresh specimen I could tell you but being exhumed and being possibly dry, dehydrated, I just

" simply don't know.

I know that the magnitude is such that I have very little doubt about the fact that this was not a maintenance dose.

Q. Doctor, could we come at it perhaps another way. If these levels had been recorded in fresh tissue would they have been consistent with the concentrations that you would expect to see in a child who had been receiving digoxin on a regular ongoing way as part of a regime of therapeutic administration?

A. No. They are much higher.

Q. They would have been higher even than that?

A. These levels are much higher than they would have been in fresh tissue in a child who had been receiving a therapeutic regime of the drug, yes. "

Dr. Hastreiter was pretty clearly of the view that, despite all the lack of experience and the difficulty

of interpreting levels in exhumed tissue, was satisfied that the order of magnitude of these numbers could not be ignored and the order of magnitude of the numbers satisfied him that they did not result from the accidental or erroneous administration of one dose to the child.

Dr. Kauffman was not willing to attach significance to the numbers, although he did acknowledge in Volume 71, page 5580 that they were "fairly high".

He was impressed by the established presence of digoxin in a child for whose death an unconfirmed cause of shunt occlusion had previously been assigned, but who had displayed symptoms of digoxin toxicity and who had had a high serum potassium level, which was also, to his mind, suggestive of digoxin toxicity.

In other words, Dr. Kauffman, although he recognized that the occluded shunt explanation may well have explained the death, in the absence of any other information that could be accepted as an explanation of the death, still left him with some question of, first, the symptoms of digoxin toxicity that the child manifested in the course of her terminal

events, but also that other thing which he regarded as symptomatic of digoxin toxicity, an elevated serum potassium level.

As between the two possible explanations of Stephanie Lombardo's death, Dr. Kauffman preferred the one, digoxin intoxication, for which, in his view, there was objective evidence, that is to say the presence of digoxin where there should have been none.

That explanation did have the benefit of objective evidence, considered the other, the occluded shunt explanation was an explanation for which there was no objective evidence.

That view of Dr. Kauffman, sir, is found at Volume 71, page 5581.

Again, Mr. Commissioner, putting that reasoning which I may say so has an attractive practicality about it, putting that reasoning together with everything that we know about this child, the events leading to her death and the circumstances on the ward surrounding her death, putting that all together suggests that here too, you may properly find that Stephanie Lombardo died of digoxin toxicity, resulting from her having received an unprescribed, and if you are prepared to accept Dr. Hastreiter's opinion,

excessive dose of digoxin.

As to whether any such dose was administered deliberately or by accident or by error, it should first be noted that the only medication prescribed for Baby Lombardo was heparin. It has been suggested rather tepidly, I think, has been suggested once or twice that there may have been a mixup between heparin and digoxin.

Nurse Ganassin, and she gave evidence, and her evidence on this point is found at Volume 140, pages 2360 to 2365, Nurse Ganassin agreed that was unlikely, particularly considering the string of mistakes that would have to be made to produce the error and Dr. McGee, when advised of the evidence as to the lack of fuss and bustle on the ward that night, the night Baby Lombardo died, agreed that given the low stress level, the sequence of errors at the time that had to be involved would not be likely. That evidence, sir, is found in Volume 146, pages 3646 to 7.

There is then, in my submission, no evidence to suggest that there was any error or accident to explain Baby Lombardo's having received digoxin.

I then come to Baby Belanger, the

last of the group of children for whom digoxin was not prescribed, but for whom was found.

Jesse Belanger is the first child on the list so far who did not go into his final decline in the middle of the night. He got into trouble at the end of the long dayshift on December 28th, 1980, and he died at 8:16 that evening. The Traynor nursing team worked the long dayshift on December 28th.

Baby Belanger was six weeks old. He had been admitted to the Hospital for Sick Children at two days of age on November 19th, 1980. His status on administration is summarized by Dr. Freedom in his reporting letter to the referring physician. It is found at page 10 of the chart. Perhaps I can read it. He quotes:

> " As you know, I catheterized this infant on the 20th of November, 1980 in order to clarify the complex congenital cardiac malformations. As you know, we were both concerned that this infant may have a serious, potentially lethal underlying chromosome abnormality but we do feel it important to establish the

" cardiac diagnosis.

The catheter study was performed uneventfully on the morning of November 20th, 1980. In brief this revealed the aorta was reasonably saturated at 93% and the angiography revealed a single left ventricle with an outlet chamber and normally connected great arteries. There was valve and subvalue pulmonary stenosis with mild to moderate hypoplasia of the main and branch pulmonary arteries. There is definitely a common atrioventricular orifice that seems connected primarily to the left ventricle and the right ventricle is represented by an outlet chamber that supports the pulmonary valve and artery. There is evidence of an atrial septal defect. There is also a right-sided aortic arch with an abberant left subclavian artery. In summary then, this infant has

There is concern about other problems, genetic problems. There are suspected chromosomal anomalies, although later those fears prove to be groundless and from page 26 of the chart it appears that the child was chromosomally normal.

Jesse Belanger's course in his first two or three weeks in the Hospital is characterized, I suggest, by stable, vital signs with increasing syanosis. By the end of November, as appears from page 44 of the chart: hypothermia has been noted and having trouble maintaining his temperature. The oxygen saturation of the blood is declining and also on page 44 by December 3rd -- I'm sorry, page 45. By December 3rd, PO2 is as low as 21 and at the bottom of the page, note appears:

> " This PO2 can't be doing his brain much good. Perhaps we should discuss early shunt provision with cardiologists rather than wait for further genetic analysis and will discuss in the morning. "

So there is now concern about oxygenation of the blood, the effect that that might be having on his general state, in particular on his brain.

On December 4th, page 49 of the chart

" complex congenital heart disease and at the present time, is quite nicely balanced with a significant degree of valve and subvalve pulmonary stenosis. Despite this however, his saturation is excellent. At the present time, there is no need for surgical intervention, but if his karyotype proves normal and if in time he would develop increasing pulmonary outflow tract obstruction (which I think quite likely) then he would be a candidate for some form of systemic to pulmonary artery shunt.

At the present time, this baby is not in heart failure, requires no cardiac medications and I think a lot rides on the chromosome analysis. "

You have got a child with complex congenital heart disease, clearly. Despite all of that apparently good oxygenation, no immediate need for surgery. He is not then in congestive heart failure and he doesn't need any cardiac medications.

Dr. Rowe wrote a note:

" The signs suggest that infundibula pulmonary stenosis may have become more significant, ie. T/F like physiology. In the event of progressive hypoxic signs propanolol would be a reasonable therapy on trial. A shunt is a likely later need. "

It was becoming pretty clear that this child was going to be a candidate for surgery. The position appeared to be deteriorating, but still no sign of enormous urgency. People were not yet talking about surgery tomorrow to get this thing fixed up.

During the first two weeks of December the baby continued to have stable vital signs, but again increasing syanosis and appears from pages 52 to 53 of the chart.

On December 20, 1980, Dr. Rowe's note on page 55, a decision is made to operate.

At the top of the page on 55:

" This baby is after discussion with the ... "

I'm not sure who that is in the family.

" ... to undergo an aorta pulmonary shunt and at present this is planned

" for 21/12/80. I have spoken to the parents about surgery as has Dr. Freedom. Dr. ... "

Somebody.

" ... and the surgical representatives. "

A decision is then made as of December 20th to take this child to surgery December 21st. You have to go right back to the beginning of the chart, page 8, to see Dr. Williams' reporting letter to Dr. Freedom on that surgery.

- - - - -

E/DM/ko

There are a couple of things of significance in the letter, in the first paragraph:

> "I operated on this baby on December 22nd to palliate his congenital heart defect. He has infundibular stenosis with VSD and likely an A-V canal. There was also some concern about the size of the right ventricle, so that he may never be suitable for total repair."

He goes on to describe other anomalies in the child's heart and vascular system.

In the second paragraph there is concern expressed about the size of the shunt, about two-thirds of the way through the paragraph:

> "On opening the shunt there was a rather modest thrill over the pulmonary artery and I was dis-appointed the artery was as long as it was. It measured about 18 mm in length and 3½-4 mm in diameter. The positioning of the shunt seemed ideal but I am concerned that it is a bit small."

And in that third paragraph he reports

on the post-operative status of the child:

> "Post-operatively he has been stable and his saturation has been hovering about 70% to 80%. His chest X-ray this morning shows definite increased vascularity on the left but we are still watching him as to whether his shunt is sufficiently large. I think that if there is much further question about it we should probably reoperate on him and do a central shunt."

So already the surgeon recognizes the possible problem with the surgery that has been performed, and recognizes the very real possibility that further surgery may be required.

On December 26th the child is transferred from the ICU to 7G, it appears there was a shortage of space in the ICU and with Dr. Rowe's blessing, no doubt reluctantly given, the child went to 7G from the ICU and at that stage he had a collapsed left lung.

On December 28th he went from 7G to Ward 4B in the early afternoon. The nursing note by Nurse Reaper at page 64 of the chart is of some interest

E 3

in following the child's progress. The top of the page it goes from 1 o'clock until 7 o'clock in the evening and she reports:

> "Child stable during afternoon apex 134 to 170 and regular. D tube fed at 1400 and retained. Suctioned for moderate amount of white mucus. Colour remained pink.
>
> 1830 - apex noted to be irregular,colour somewhat dusky. Respirations up to 80 and very shallow. D tube feeding in progress, position checked twice. Suctioned orally for moderate amount white mucus. Colour extremely poor. Doctor notified and present. Apex dropped and cardiac arrest called."

From the time of the transfer to the fourth floor Baby Belanger from a review of the physician's orders and medication sheet does not appear to have been on any medications, he had never been on digoxin.

At autopsy, as appears from page 22 of the chart the shunt was found to be patent.

Most of the physicians from whom we have heard considered Baby Belanger's death to be consistent

E 4

with his clinical condition. Dr. Hastreiter gave him a severity rating of 8. Dr. Kauffman considered that the baby by virtue of his cardiac condition was at a high risk of dying, and indeed was predisposed to a sudden deterioration. In my submission the deterioration recorded on the day of this child's death could reasonably be described as a sudden deterioration.

Now again, many months later, Baby Belanger's body was exhumed. The tissues were taken at autopsy of that exhumed body and it was sent to Mr. Cimbura at the Centre of Forensic Sciences for digoxin analysis. The results are set out in Exhibit 95E at pages 2 to 3 and they disclose that in the exhumed liver sample there was a concentration of 253 nanograms per gram of digoxin, that is to say after an analysis by HPLC/RIA, and after the same kind of analysis a concentration of 43 nanograms per gram in muscle tissue.

You will recall, sir, that Belanger was one of the two children where identification of digoxin was confirmed by gas chromotography and mass spectometry. In recognizing all of the difficulties of interpreting levels found in exhumed tissue I note merely that the liver concentration recorded in this child is above the levels recorded in fresh livers of infants on digoxin

E 5

therapy.

Now clearly those toxicological findings again raised a whole new set of questions about Baby Belanger. First and foremost was it the digoxin that caused his death? Everyone agrees here as with Lombardo that the death and the manner of dying of this child were consistent with digoxin intoxication.

Dr. Kauffman's evidence found at Volume 71, page 5622; Dr. Kauffman considered the terminal events described in the chart to be more consistent with digoxin intoxication than with the baby's clinical condition, but again the question is whether digoxin intoxication caused the child's death and the elements that in my submission go into that determination are these:

Digoxin was found where none should have been found and I suggest it is a reasonable inference that digoxin found its way into that child's body by being administered to him during life, no other explanation has been offered by anyone.

Second, although the only sure significance of the digoxin finding is qualitative it has to be asked, I suggest, whether anything can be taken quantitatively from the recorded levels, one of which is in the toxic range of concentrations for fresh tissue.

E 6

Now Dr. Hastreiter considered the toxicologic data along with the clinical data and concluded that digoxin toxicity was probably the cause of death. He did not believe that the digoxin concentrations of the order reported could be produced by a single maintenance dose, or even a single loading dose given in error. He also considered the sudden and in his view unexpected nature of the terminal events, and he concluded that Baby Belanger probably died of digoxin intoxication resulting from a deliberately administered overdose of the drug. That evidence, sir, is found at Volume 77, pages 6798 to 6809.

Dr. Kauffman, although he acknowledges that digoxin toxicity was not and could not be proved, gave us his opinion that there was a high probability of digoxin toxicity and that digoxin at least contributed to Baby Belanger's death and he thought it unlikely that the death had been caused by a medication error. That is found in Volume 73, pages 6068 to 6069.

In my submission in order to make those statements Dr. Kauffman must, to some extent at least be attaching some significance to the quantitative findings, not merely to the qualitative findings.

In summary, Mr. Commissioner, you have a

E 7

child here who died in the early evening having gone into sudden decline in the presence of members of a particular nursing team. Who although very sick with a not good prognosis may not have been expected to die just when he did. A child for whose sudden death there is no immedicate clinical explanation, other than the proposition that his cardiac status put him at risk of death and could have caused him to deteriorate and die suddenly. Certainly, however, death was not the result of a sudden occlusion of his shunt. We have a baby whose terminal symptoms are consistent with his clinical status and are also consistent, or in Dr. Kauffman's view more consistent with digoxin toxicity, we have a child in whose exhumed body digoxin was found in concentrations which were they in fresh tissue would be indicative of toxicity, although this was a child for whom the drug was never prescribed.

In all of the circumstances, in my submission, you are entitled to infer and find that Jesse Belanger probably died of digoxin toxicity resulting from the unprescribed administration of digoxin in a toxic sized dose and that there is no evidence to suggest and Drs. Hastreiter, Mirkin and Kauffman think it unlikely that digoxin was administered to this baby by error.

E 8

Now Mr. Commissioner, those are the three other children then for whom digoxin was not prescribed but in whom it was found and I have made the same submission with respect to each of them to you.

As I told you, I want to move now to that group of five patients in respect of whom "Do not resuscitate" orders were written. I propose to do that really for two reasons. First, although as I said on Thursday every case has to be reviewed to see if there are events or circumstances which excite suspicion, and the severity of a child's condition or the obliqueness of his prognosis don't by any means guarantee that his death when it occurred was a natural one. It has nevertheless to be acknowledged that the hopelessly and clearly terminally sick child is likely in the circumstances of our situation to be found to have died a natural death. I say that because for so many of these children there is no clear toxicological evidence, but when there is clear compelling compatibility between the death and the irreversible clinical condition it is very difficult to suggest that something other than clinical condition may have caused the death.

If there was a killer on the cardiology wards, as in my submission there was, then a review of

E 9 all of the cases that we have to consider does not suggest that children were selected for death because they were terminally ill. Certainly some of the children about whom I have already made submissions and some of those about whom I shall be making submissions, were extremely sick. But it is difficult to explain rationally why perhaps a misplaced tenderness for such children and the desire that they not suffer further would lead anyone to kill, for example, Justin Cook on the eve of surgery which might have saved him. Other children whose deaths I will suggest would have been at least highly suspicious, were far from hopelessly sick. Baby Velasquez, for example, was recovering from surgery and was expected to be sent home in the near future. In short, if one is entitled to consider whether a motive existed as an aid to determine how children died, it is my submission that mercy killing or euthanasia is not a strong candidate. Indeed it would be my submission that none of those children who were so mortally sick that no resuscitation effort was to be made, none of them died in circumstances to give rise to suspicion. That indeed if it be so may be significant. For some reason, if I am correct, those for whom a Code 25 was not to be called, although exhypothesis they were

E 10 therefore desperately and terminally ill, those for whom a Code 25 was not to be called appear not to have attracted the attention of whomever it was, according to my thesis, who was deliberately administering overdoses of digoxin to children.

- - - -

You will recall, sir, that three of the "do not resuscitate" patients were older children and for them their age and ability to relate what someone had done would no doubt militate against any interference with them. But two were infants and it would be my submission that they too died natural deaths.

My second reason for turning to this group of patients at this point in my submission is to consider the manner of their dying and to compare and contrast it with the way in which so many of the other children died. Just as other patterns and common threads, each in and of itself ambiguous, may when taken in the round assume a significance that would be helpful to you, so too, I suggest, are the repeated pattern of death and terminal symptoms although ambiguous if considered alone may add to the composite picture that you have to have of these children.

I will deal with the group if I may in chronological order of their deaths.

THE COMMISSIONER: I understand that Murphy, Floryn and Heyworth are the three older children.

MR. LAMEK: We have Perreault, Murphy,

Heyworth, and Leith and Perreault and Leith were the infants.

Alan Perreault died at 1:45 in the afternoon of July 8th on ward 4A. The Trayner team was on duty. Indeed we have heard from Mrs. Trayner that this baby died in her arms. Baby Perreault had that congenital heart defect known as hypoplastic left heart syndrome.

In Volume 12 beginning at page 2088, Dr. Rowe discussed the baby's heart defect and told us that babies with that particular syndrome died at an average age of 4½ days as soon as the ductus arteriosis closes. They are dependent upon the ductus for getting the blood to the lungs. He says that only in exceptional cases do babies with this condition live much longer than a week. Beyond question, Alan Perreault was not going to survive. The amazing thing about him was that he lived for 26 or 27 days. Nothing could be done for him although so surprising was his survival that the cardiologists went back to be sure of their diagnosis and they even contemplated the possibility of experimental heroic surgery before he at last died. His course is summed up in the death report at page 30 of the chart. It says that he was referred from the North

York Hospital at the age of 12 days and he had gross congestive heart failure. He was dusky, had a fast respiratory rate, an enlarged liver, poor peripheral pulses, gallop rhythm, soft systolic murmur. It cites the investigations that were done and then:

> " In view of the diagnosis of hypoplastic left heart and its inevitably poor outcome, no active medical treatment was undertaken with full consent of both parents. The baby gradually deteriorated and died at 1345 hours on 8.7.80. A limited post mortem examination of heart and lungs will be performed. "

The baby's course from July 5th to July 8th as it appears in the nurses' and physicians' notes in the chart beginning at page 40 and following, is I suggest one of ongoing deterioration. Respirations were slowly and progressively becoming more difficult for this child. There are signs of worsening congestive heart failure with increasingly puffy feet and extremities. His colour is worsening as the days go by. He developed a gallop heart rhythm -

becomes more frequent.

By the long dayshift of July 8th, it was expected, according to Mrs. Trayner, that he would die that day. That is in her evidence in Volume 137, page 1501. She says that they were told by the physicians to expect the baby to die that day.

Dr. Rowe in reviewing the events leading to the child's death said this at Volume 12, page 2099, beginning at line 10, in direct examination by me:

> " Q. Doctor, I have to put to you that that death is in very sharp contrast to the sequences that we were referring repeatedly yesterday, was it not?
>
> A. Yes.
>
> Q. Now, here was a baby who was certainly very seriously sick, and inevitably would die as you have said. I asked you yesterday whether the pattern of terminal events that we were seeing was common, and I don't suggest for a moment that one can base anything

" upon this one child, but nevertheless here is one baby who certainly fits the bill of a very, very sick child. The manner of his death was entirely, entirely different from anything that we have looked at so far. Is that a matter of any significance in the exercise on which we are engaged, Doctor?

A. No, I don't believe so. He had been in - despite the suggestion that he had been stable, he had been in chronic congestive failure. The huge surprise of course had been that he hadn't died before.

Q. Yes.

A. That he had signs of gallop rhythm and bad congestive failure, his liver was six centimetres below the custom margin so he was obviously getting worse.

Q. Yes.

A. Getting worse over time so his condition was really not stable. I think if you compare the notes from

" the 5th of July to the 7th and they are written by a cardiologist, the notes I am referring, there is a change.

Q. I don't suggest he was stable, Doctor, indeed the pattern is one of slow and steady decline.

A. Deterioration.

Q. Until fatal entirely.

A. Yes.

Q. In very sharp contrast to what we were seeing yesterday. That is quite often a pattern of stability and then a very sudden and rapid decline with dramatic terminal event.

A. Yes. "

Dr. Rowe agreed that there was no evidence of any arrhythmia here although he assumed that Baby Perreault's heart probably slowed before he actually died. In terms of arrhythmia he agreed that there was no evidence in the chart to suggest any rhythm disturbance as we have seen in other cases, no suggestion of any seizure activity or anything dramatic of that kind. He did suggest that there was some event that occurred when the baby began to exhibit Cheyne Stokes breathing

and the rate of deterioration increased.

Mrs. Trayner who was present at the end gave this evidence, Volume 129, page 85 beginning at line 20:

" Q. Forgive me, I have no doubt it is still a very unpleasant memory. Can you describe for me what happened with this child? Did he - can I put it this way, as between extremes, did he sort of drift away or did he suddenly die?

A. Well, to use your words, he drifted.

Q. Those are very imprecise words I understand, but you catch the meaning, the distinction I am trying to draw?

A. Yes.

Q. When you say he was dying, you are conjuring up a picture that we have of someone dying, sort of getting weaker and fainter and fainter and finally dying.

A. Right.

" Q. And is that your recollection of what happened with this child?

A. Yes, it was. "

There were no recorded symptoms of digoxin toxicity with Alan Perreault. The nurse who was there holding the child as he died agreed that there was no dramatic event about his death, the child drifted away. Every physician who opined in this death said that the death was entirely consistent with the severe clinical condition of the baby. Dr. Hastreiter scored him 10 on the severity rating - the most severe rating. Every physician said that there was nothing about the death that was indicative of digoxin intoxication. Everyone expressed the opinion that the death was a natural death.

In my submission there is nothing either in the objective evidence or in the medical opinion evidence to suggest that there was anything suspicious about the death of Alan Perreault.

- - - - -

G
RD/cr

---On resuming.

THE COMMISSIONER: Yes, Mr. Lamek.

MR. LAMEK: The next child in chronological sequence of those for whom there was a DNR order was Paul Murphy. He died at 10:25 in the evening on August 23rd, on Ward 4A. Mrs. Trayner's team was on duty.

This was a 15 year old boy, sir, who spent his life in and out of the Hospital for Sick Children and by the summer of 1980 sadly he was clearly dying.

At page 324 of the chart, Dr. Fowler's letter of July 16, 1980 to his referring physician, Dr. Dennis in Brampton. He had seen Paul Murphy on July 15th, the day before for re-assessment. He recites the history there following of this boy. The second sentence of the first paragraph:

> "He has had surgery but a haemodynamic study post-operatively explained his persistent intractible heart failure on the basis of a very small pulmonary arteries with a poor functioning right ventricle. He was admitted to Hospital about three weeks ago with a severe

"electrolyte problem and mental confusion. When he had his electrolytes stabilized he returned to his usual mental status and was eventually sent home about a week ago. Since that time he has been vomiting 2-3 times a day, except for the most recent week-end when he did not vomit. In the last two days, however, he has been vomiting again and the parents have noted intermittent mental confusion. He has been mildly cyanosed and there has been a moderate degree of swelling of his ankles, particularly in the evening."

Plainly the congestive heart failure, which had plagued this boy for years, was becoming progressively worse.

Then "Conclusions and Recommendations" at the bottom of the page:

"On the basis of this assessment I feel that this boy is again having a lot of difficulty because of his severe intractible heart failure, secondary right ventricular disease

"and small pulmonary arteries. He was discharged on digoxin and Hydrodiuril... Because of the gravity of the situation I felt that he should be admitted for further stabilization. He was intermittently rather confused during my examination, but most of the time was lucid.

This boy is remarkable in that he seems to come around after his many difficulties. I feel that he probably is not going to survive long, but I am sure we can make him more comfortable by changing his diuretic regimen and increasing his serum potassium level."

On September 12th at page 17 of the chart Dr. Fowler wrote again to Dr. Dennis following the boy's death:

"This boy was admitted to Hospital for adjustment of his medications and died.

I am sorry about the outcome but I think in the last few months it was inevitable that he would not survive because of his high pulmonary pressure

"and poor right ventricular function." Plainly, by the summer of 1980 this child, young man, had progressive and irreversible congestive heart failure and was not expected to survive.

Over the last couple of days of his life there are described in the nursing note, particularly at page 127 to 131 of the chart:

> "Symptoms were becoming more marked and there was a generalized swelling and puffiness indicating retention of fluids. He was dis-oriented and he was having difficulty breathing. He was appearing drowsy. His mother was noticing behavioural changes."

When we come to page 131 of the chart there is a final nursing note from Miss Cooney:

> "1930 to 2200, vital signs - stable. Behaviour - orientated lapsing into confusion in later evening. Taking sips of water, requesting oxygen off and on. 2200 - patient sitting up in bed - very confused. 2210 - patient involuntary of stool.
>
> 2215 - patient rolled down and turned to side - then became unresponsive -

"respiration at this time very shallow and laboured - blood pressure hard to obtain. Oxygen given 40 per cent by mask. Dr. Wilkinson called."

Thirty minutes later the child was pronounced dead.

Dr. Wilkinson's note begins at the bottom of page 130:

"Called to see Paul, because of lack of responsiveness. When examined he had no detectable blood pressure, pulses, heart beat or respirations. Clearance of airway, stimulation and oxygen did not have any beneficial result.

Paul had been noted to be sitting up; talked to his nurses and seemed well oriented just minutes before. No evidence of vomiting, aspiration. Had not been eating or drinking anything. Patient was pronounced dead at 10:28 p.m."

Again, the unanimous view of the physicians was that the death of Paul Murphy was entirely consistent with his clinical condition. Dr. Hastreiter scored him a 9 on the severity rating.

Although Dr. Hastreiter felt that the terminal event had been somewhat sudden and unexpected he agreed that the death was a natural one. That evidence is found in Volume 81, page 7508 and certainly there were no symptoms, that I at least would recognize, as being those of digoxin intoxication recorded in the chart. There is no evidence of any sudden onset of bradycardia or vomiting or anything of that sort.

Dr. Rowe presumed or inferred that at the very end a major arryhthmia was experienced which caused the heart to stop and he said that the death, when it occurred, was a sudden one, as it indeed appears to have been.

In my submission, the matter of Paul Murphy's decline and death is very different from that which appears in many, many other cases. He declined to the point of death over a number of days and, in my submission, there is no reason to challenge the unanimous medical opinion that Paul Murphy died a natural death brought about by his cardiac status and clinical condition.

Laurette Heyworth, too, was an older child. She was 11 years old when she died on September 2nd, 1980. She died at 8:30 in the morning in Ward 4A and the members of Mrs Trayner's team

were on duty on 4A at that time. Like Paul Murphy Laurette Heyworth was not a stranger to the Hospital for Sick Children. The clinical condition was serious, very serious and she, too, had received a severity scoring of 9 from Dr. Hastreiter. Indeed, Mr. Commissioner, her prognosis was as gloomy as it could be. She had come to the end of her life and nothing could be done for her.

On August 27th, though, a new symptom appeared, she vomited twice. The digoxin level taken that day was reported back with a level of 2.5 nanograms per millilitre. It was suspected that she might be showing some symptoms of digoxin toxicity and digoxin was, therefore, ordered held. That order appears at page 180 of the chart and at page 153 in the notes there is reference to it.

There was further vomiting on August 28th, the next day and digoxin was then ordered restarted and then re-ordered held again.

It appears that digoxin and other medications were kept on hold on August 29th until tomorrow morning. On August 29th, as appears from page 183 of the chart, digoxin was restarted, but to be administered intravenously. The order there appears on page 183 under date 29/8/1980 calling for

lasix IV and digoxin IV twice a day.

According to the medication sheet at page 187 digoxin was administered on the 29th twice by Dr. Sturm. On September 30th the order was changed again and it appears at page 184, to an oral dose of digoxin. I am sorry, that is August 30th. It seems from the medication sheet that the drug was not administered on the 30th.

On page 188 it appears to have been administered by nurses on the 31st and the 1st of September: oral administration.

- - - - -

H-1
DM/ac

So there is a rather complicated history of digoxin orders for this child in the last two or three days of her life. Again, sir, I suggest the picture is one of steady decline. Page 165 of the chart, the nursing notes, it appears that during her last night she was cyanosed when not on oxygen, she had difficulty breathing, she was very jaundiced, complaining of thirst and dry mouth, severe swelling in her lower legs, abdominal pain, she was extremely restless. At 7:30 in the morning it is recorded that she was complaining of shortness of breath, she was on oxygen and very uncomfortable. At 8:30 in the morning she died.

In my submission the chart discloses no symptoms of digoxin poisoning during the last hours of her life. Dr. Rowe suggested that the only symptom that might be indicative of digoxin toxicity was the recording of several ectopic heart beats since about midnight. Certainly there is no sign here of any acute episode of intoxication. There is no sudden change in her condition, no abrupt move from anything that looked like relative stability to the direst emergency. In my submission again this is simply not a pattern of death that was manifested in so many of the other cases. I

suggest, sir, that there is no reason to suspect that Laurette Heyworth died of anything but a natural death brought relentlessly on by her irreversible clinical condition.

Bruce Floryn was the third of the older children for whom a "do not resuscitate" order was in place. He was 19 years old when he died. He died on ward 4B at 6:20 of the morning of February the 7th. At that time there were members of the Trayner team on duty on ward 4A. Like Paul Murphy and Laurette Heyworth, Bruce Floryn had been in the Hospital for Sick Children several times and indeed consideration was given to him as a candidate for heart transplant but he was not considered a good candidate and that idea was abandoned. His condition was very serious and he was not going to get better. Nothing could be done for Bruce Floryn. Dr. Hastreiter gave him a severity rating of 9. Clearly he had chronic, severe and worsening congestive heart failure and by the time of his final admission on January 27th, 1981, he had a host of other problems too.

His history, the ailments, and his course during his final hospital admission are summarized in the final autopsy report and in particular

pages 59 and 60 of the chart, and the narrative portion is on page 60. It is recorded:

> " ... presented with congestive heart failure secondary to an idiopathic congestive cardiomyopathy. He had congenital AV block and received a cardiac pacemaker in 1974. Severe congestive heart failure developed over the following few years. He had several admissions for medical management and was considered for cardiac transplantation but was rejected on the basis of very poor haemodynamic status. His recent medical therapy included digoxin, lasix, aldactone, Minipress, ASA, mycostatin. He presented this admission with increasing weight gain and edema in spite of increased dosage of his various medications. "

Indications as I understand it that CHF, congestive heart failure, was not being controlled.

> " Other problems on admission included hyponatremia ... "

Which I understand to be too little sodium, I think:

> " ... and multiple chronic stasis skin ulcers. Following admission the patient initially improved. Four days after, however, he developed purpura on his abdominal wall possibly secondary to chronic liver disease. Subsequently he began to deteriorate in spite of medical therapy with increasing weight gain, increasing edema and intermittent episodes of disorientation and confusion. His condition progressively worsened. An additional complaint noted just prior to death was severe chest pain and pain on swallowing. He died on the 11th day following admission. "

There is then set out the findings at autopsy and the final sentence of that second paragraph on page 60:

> " Death in this case is attributed to severe congestive heart failure,

" with contributing factors of multiple pulmonary thromo-emboli and infarction. "

It then goes on to list further pathological findings in this child.

It appears from the notes at page 102 of the chart that the morning before he died Bruce Floryn was very disoriented, he got out of bed, he fell, he was uncomfortable during the night and could not get comfortable. All observations that are reminiscent of the chart of Laurette Heyworth that we looked at a few moments ago. Page 103 of the chart, during the day of February 6th, Bruce Floryn's father noted that his respirations had become shallower and that after administration of a Brompton cocktail, a sedative and analgesic. In the evening he was very unresponsive and limp and his blood pressure was down. There was increased cyanosis in his extremities and his respirations at one point were noted at being down to 16 per minute.

Page 104 of the chart Dr. Runge wrote a note at 11:00 in the evening on February the 6th and recorded:

" Reviewed patient secondary to (1) no

" urine output for 11½ hours. (2)
Very drowsy since receiving Brompton's cocktail. Responds appropriately to painful stimuli, rouseable. Quickly falls back to sleep after responding with 1 or 2 words answer to simple questions. Has difficulty maintaining body temperature.
Pulse 60/min poor volume, regular.
Chest - good, air entry anteriorly, no creps.
Liver extended 6 cm. at right costal margin.
Impression: progressive end stage cardiac failure. "

Page 105, the nursing note, this is 6.2.81:

" At 2230 has not voided since 1200 hours.
Noted congested cough occasionally. Respiration shallow difficult to rouse. Very brief periods of consciousness, unable to complete sentences.
Respirations remain shallow. "

This is 7.2.81, at 2:00 in the morning:

" Lips are cyanosed. Extremities cyanosed. Basically unchanged mental state. No extensive periods of orientation noted. Remains very difficult to rouse. Very weak. "

His apex is unchanged, then at 6:10 on the morning of the 7th a sudden marked change in respirations, increased cyanosis, unresponsive, very shallow, weak, ineffective respirations, and Dr. Runge comes and he writes the note set out at the bottom of page 105. Upon arrival he finds no pulse, respirations undetectable, pupils fixed and dilated. The patient is pronouced dead at 6:30.

That, Mr. Commissioner, in my submission is a terribly sad picture of a young man finally succumbing to irreversible and progressively more severe congestive heart failure.

Now at 6:10, as I said, Nurse Bracewell noted a sudden marked change in respirations. Perhaps I would remind you respectfully of just what it was she said:

" Sudden marked change in respirations. Increased cyanosis. Unresponsive. Very shallow, weak, ineffective

" respirations. "

And with respect I suggest that sounds not so much as a great change in the nature of his breathing, he had had shallow breathing for 24 hours, but it was clearly a change in the degree of shallowness. Again, there is nothing noted of the kind that we have learned over the course of the last year to recognize as symptomatic of digoxin intoxication. I suggest the picture is of a patient who is loosening his hold over life over an extended period of time.

The unanimous medical opinion was that death was not only consistent with but was caused by his clinical condition and that there was no indication of digoxin toxicity. I invite you respectfully, sir, to make those findings with respect to Bruce Floryn.

- - - - -

I/DP/ko

The last of these "Do Not Resuscitate" patients is David Leith. He died at 10:30 in the morning on March 6th, 1981. He was six weeks old. His condition had worsened during the night. The Trayner nursing team was not on duty for the long night of March 5 to 6 and one member of the team only was on duty for the long day shift of March 6th when the child actually died.

On February 2nd, David Leith had had a surgical repair of a coarctation of the aorta but he had other severe cardiac problems including hypoplastic left heart syndrome and he had severe congestive heart failure. He had a history of arrhythmias. I refer, for example, to the long day nursing note for March 2, page 170 of the chart, and it is recorded "Apex ranging from 140-152 mostly slightly irregular to more irregular towards end of shift". Bounding type of heart rate. Page 174, during the long day shift of March 3rd, it is noted the apex of 138-159 was one episode of irregularity. At the bottom of that page, 1900 hours, vital signs, apex 137 to 149 and five episodes of irregularities. That is on March 4.

David Leith's parents were aware that their child was not going to make it. Page 175, at the top, the long day of March 4, conversation is

recorded, what the parents stated. The mother stated that it was difficult knowing he was not going to make it. The nursing note on the long day of March 5th, page 176, apex 150 to 142, two episodes of irregularity.

At the bottom of page 176, sir, there is a note which I believe to be written by Dr. Mangera recording a visit, and review of the child, on March 5 at 8:15 in the evening. On page 177, a further note I believe from Dr. Mangera. On the 5th she recorded urine output reduced, weight was up, fast respiration and distress and the liver was enlarged, extending to 3 centimetres.

On the 6th the note is "Worse. Tachypneic, cyanosed, distressed." Respiratory rate, 60 to 70, indrawing, cyanosed, urinary output down, question mark pulmonary edema, proposes lasix, chest X-ray - massive oversized heart. Then at the bottom "hold next dose of dig.". I will leave it at that. I am not exactly clear on my reading of the evidence about this child.

Page 178, the cardiology fellow has a note "CHF worsened, severe (something). Lasix twice produced a poor response. Pulmonary edema and massive cardiomegaly. The mother was informed."

The final nursing note found on page 178

I 3 in the lower half of the page from 7:30 in the evening of the 6th until 7:15 in the morning - respiration more than 100 per minute throughout the night. At 4:20 in the prone position at the end of naso-gastric tube, respiration rate down to 50, then down to 40 at 4:30. Air hungry and deep indrawing. Colour remained very pale and dusky. Dr. Mangera was called. Child was given lasix. IV initiated by Dr. Mangera. Following lasix voided 5 cc of urine. Very restless - given morphine; given sodium bicarb; given phenobarb. At 7:30, at the bottom of the page, respiratory rate goes up to 126 minutes, apex is stable and the colour very slightly improved. The note on page 179, at 10:35 in the morning of the 6th, babe in mother's arms, 60 percent oxygen, colour pasty gray, gasping respirations from 60 to 28 to zero. Apex 128 down to 100, down to 60 to zero. Blood pressure from 80 over pulse at beginning of shift, went down to 40 over pulse. Babe became more agitated. Morphine was given at about 9:30. The patient's monitor showed that his heart stopped at 10:25. No irregularity noted there, slowing certainly but not irregularity, no arrhythmias, no arrhythmias noted of any kind. Again in my submission it is a picture of a small terribly sick child who faded away and died. All of the physicians

I 4 for the Hospital for Sick Children and outside agreed this was a natural death. In my submission there is nothing in the chart or in the evidence which provides any sort of a basis for dispute over that conclusion. I invite you to make a finding that is consistent with the unanimous opinion of the medical experts.

Mr. Commissioner, those are the patients for whom no resuscitation was attempted and having now reviewed each of those cases I make the observation to which I earlier referred. In none of those five cases is there any suggestion or evidence that digoxin toxicity may have played a part in causing death. In my submission you should find that each of these children died from natural causes and that no suspicion attaches to any of those five deaths. If that be so, and I say again one has to wonder whether mercy killing of which we heard a good deal during the course of the evidence is an acceptable explanation for the deaths of other children. Those other children I have already made submissions about and suggested that they were not killed. About others for whom there is serious reason to think that they may have been killed by deliberate overdoses, if euthanasia was a motive one has to wonder why Babies Perreault and Leith were allowed to linger on until their inevitable deaths

I 5

occurred naturally.

THE COMMISSIONER: I am just wondering about that. Is it not possible that when the children were dying that there was no need for mercy killing? Mercy killing is in a sense killing to speed up the deaths, or if mercy killing was to save from an unsatisfactory life, it might be different. I don't know.

MR. LAMEK: I suppose my only difficulty with that proposition, Mr. Commissioner, is that there are children for whom surgery was planned, Cook, and of whom it would be my submission that he was killed, it is difficult to understand why in an attempt to avoid suffering there would be contemplation of killing a child for whom surgery was planned by which he might thereby be improved and not elect to shorten the life of someone who is irreversibly sick and clearly in very great difficulty.

Secondly, Mr. Commissioner, the way in which these children came, in my submission, to a perfectly natural death is in very stark contrast to the pattern of terminal events to which I have already referred in children already discussed and to which I will refer again often, I do not for a moment presume to challenge Dr. Rowe's evidence that any

I 6

pediatric cardiac patient can go into an irreversible rapid decline in the blinking of an eye. I do not challenge that that can happen in any of these children. I have no doubt that it can.

Respectfully I point out two things. First, the nurses, and I am not pitting their experience against that of Dr. Rowe, but the nurses were clearly troubled by the fact that patients who appeared relatively stable suddenly went sour and died. Second, we have just reviewed five patients including two infants who in my submission clearly died natural deaths. Not one of them displayed that pattern of terminal events, a sudden precipitous decline from relative stability. No doubt it can occur in any pediatric patient. In my submission it did not occur in these five natural deaths.

Mr. Commission, I propose to turn now to a group of four babies for whom there are toxicological data which may be suggestive of digoxin toxicity having played a part in their deaths. Again I propose to deal with them chronologically in the order of their deaths. They are Babies Thomas, Gionas, Inwood and Gardner.

Jennifer Thomas died in Room 418 on Ward 4A at 3:38 in the morning of February 12th, 1981.

I 7

When she was nine days old she was admitted to hospital on February 10th. She suffered an onset of critical symptoms at 3 o'clock on that morning and that led swiftly to cardiac arrest and death. Mrs. Trayner's team was on duty on Ward 4A for the long night shift of February 11 to 12.

Baby Thomas was scheduled to go to surgery for a bypass operation later in the morning of February 12. She did not of course survive to reach surgery.

Dr. Rowe in Volume 16 at page 2770 described the surgery which was planned as heroic emergency surgery. This was no routine operative intervention that they had planned. This was a very, very sick child, another hypoplastic left heart syndrome case. Dr. Hastreiter gave her a severity rating of 10. Dr. Nadas, reviewing the case for the CDC, rated her condition on admission as intermediate but her prognosis as poor. She was being treated with digoxin and diuretics for congestive heart failure and with prostaglandin to keep the ductus open until she could get to the OR. Unfortunately, she developed secondary effects from the prostaglandin, affected her body temperature and the dosage or more precisely the rate of infusion had to be reduced. The physicians

I 8 were in the classic 'no win' situation. She needed the prostaglandin to keep the ductus open but was suffering side effects from the prostaglandin.

- - - -

If we turn to the chart, sir, and to the nursing note by Nurse Scott for the long nightshift of February 11th to 12th is found at page 78. It records that her apex - 180 to 140 was regular until 0300 hours. Her temperature was high. That was the effects of prostaglandin. The respiration was very regular and shallow, ranging from 96 to 82. Blood pressure between 80 and 76 over pulse. Left hand and right leg extremely cyanotic and cool to the touch. She was feeding eagerly, taking a total of 33 cc. Similac each feed and she had two feedings, tolerated feedings. An hour and a half after 11 p.m. feed, she vomited approximately 20 ccs. mostly mucus and an order was written that she was to be nothing by mouth. She was going to surgery. Elimination satisfactory in the prostaglandin. She, halfway through that note on the prostaglandin, the IV PGE rate was reduced to 1 cc. per hour due to high temperature.

So we have the child, she vomited at about 12:30, half an hour after midnight. There is no prior note of vomiting in the chart that I have seen, sir. 3:00 in the morning there is a very sudden change. The apex which had been regular, ranging from 140 to 180, now drops below 140 and

becomes irregular and proceeds quickly, according to the note, to what Nurse Scott describes as atrial flutter. CPR was started and a Code 25 was called and an unsuccessful resuscitation effort took place.

On page 77, the opposite page, sir, is Dr. Heilbut's note. He records that Jennifer apparently developed irregular rhythm and bradycardia at 3:10 a.m. and went into ventricular fibrillation. She appears to describe as ventricular fibrillation what Nurse Scott had called atrial flutter.

All of the physicians who have commented on this child, including Dr. Hastreiter and the CDC, view her death as consistent with her clinical condition, sir. Dr. Rowe's evidence is particularly interesting on the cause of death and found in Volume 16, page 2771, beginning at the bottom of the page where he said, having described the surgery that was proposed for the day she died, at line 23:

> " Part of the problem was that the baby developed secondary effects of the prostaglandins which had to be reduced in dosage, and when the dosage has to be reduced there

" is a risk that the ductus will begin to close, and if that happens everything starts to deteriorate. I would think that it is conceivable that that was the way in which this baby proceeded and the terminal events would be related to that. "

Then again at page 2777, I was asking Dr. Rowe about the terminal events and symptoms described in the chart and beginning at line 10, and then Dr. Heilbut's note on page 77, the lower half of the page:

Jennifer apparently developed irregular rhythm and bradycardia at 3:10 in the morning. Went into ventricular fibrillation and was therefore defibrillated. Routine cardiac resuscitative measures were instituted but to no avail. Time of death 3:38 a.m.

It is almost a pattern we have come to recognize, those terminal events, isn't it, Doctor?

A. Yes. It is.

Q. Very sudden onset of them?

" A. Yes.

Q. Very rapid course. Vomiting at 12:30 in the morning, arrhythmias, bradycardia to ventricular fibrillation.

Are those events, their onset and their course consistent in your judgement with digoxin intoxication, Doctor?

A. Yes. They are.

Q. Are they consistent in your judgement with the child's anatomical and clinical condition?

A. Yes. They are.

Q. Does one have to posit a closing or a constricting of the ductus in order to find that consistency?

A. Yes. "

Death and the manner of dying, as I understood Dr. Rowe, were consistent with the clinical condition if the ductus closed or became constricted.

I take you from there, sir, to page 34 of the chart, the final autopsy report.

For reasons that aren't exactly clear, page 1 of the final autopsy report is found at page 50.

Page numbered 34 is, in fact, page 2 of the autopsy report.

In the final paragraph, reporting on the findings of autopsy, it is said halfway through the paragraph:

" The ductus was patent, 7 mm in diameter. "

Now, I put that finding to Dr. Rowe and at page 2773 of the same volume 16, the following exchange took place beginning at line 7:

" MR. LAMEK: Q. Doctor, at autopsy it was discovered that the ductus had an opening of 7 millimetres. Can you tell me whether that represents some closing or whether the ductus was still fully patent? That is at page 50.

A. Yes. Well, I think that there is always a discrepancy between the anatomic findings and the functional findings.

There is a substantial amount of work in that regard experimentally and it shows that you can close the ductus so that no blood gets

" through it and yet at autopsy (and I am talking about animal work) the ductus is moderately widely open so this is related perhaps in part to the age of the patient and the way in which they die. "

As I understand Dr. Rowe, his evidence comes to this that notwithstanding the autopsy finding, as stated in the autopsy report, there may have been a sufficent constriction of the ductus to reduce the blood flow through it below the point needed by the child to secure reasonable oxygenation of her blood. If that happened it would explain her death and manner of dying. And I accept that, of course, as an honestly held opinion and one that is entitled to considerable respect, because of its source, but it is, however, an informed conjecture as to what may have happened to cause Jennifer Thomas' sudden deterioration, arrest and death.

It may be a perfectly valid conjecture and happily I am obliged to say that it is what it is, but there are several features of the death that may be ground for suspicion, that some other cause may have intervened.

In the first place, there are what I call the circumstantial elements, the pattern, the common thread elements, death in the middle of the night and the presence of members of the same nursing team, a period of apparent cardiac stability, vomiting at 12:30, at 3:00 o'clock in the morning, the sudden appearance of bradycardia, irregularities, ventricular fibrillation or atrial flutter. The cardiac function at 3:00 o'clock in the morning goes totally awry and a resuscitation effort that is to no avail.

There are also digoxin findings in fixed tissues which may, taken with everything else be of some significance. That is found at Exhibit 95A, sir, pages 9 to 10.

In fixed heart tissue there were concentrations measured in the left ventricle of 55 nanograms per gram of digoxin. In the left atrium 63 nanograms of digoxin and digoxin-like substances and in the septum 15 nanograms of digoxin.

Mr. Cimbura notes those concentrations were likely high before the tissues were put into Klotz solution.

If you turn to page 4 of Exhibit 95A, sir, you will note no. 3 which is a note following the recital of the levels found in the Cook tissues.

J-8

You will see a note to the effect that in fresh tissues the range of concentrations in ventricle of infant's on digoxin therapy, as Baby Thomas was, runs or ran as then reported in the literature, from 49 to 975 nanograms and the range in cases of fatal poisoning, ran from 108 to 1240 nanograms, a vast area of overlay.

Therefore, although the concentrations in Baby Thomas' fresh heart tissue may well have been higher than those found in the fixed tissues, one cannot say how much higher, if at all, and the levels, as they are recorded, fall squarely within the therapeutic range of concentrations in fresh tissue.

On page 10 of Exhibit 95A, sir, there is the recording of a level in fixed lung tissue, a level of 45 nanograms per gram of digoxin. That may have been higher before the tissue was fixed, but even as it stood it was beyond the therapeutic range of concentrations recorded in the literature, as noted by Mr. Cimbura on page 4, note 4 and well into the fatal poisoning range of concentrations in lung tissue, in fresh lung tissue as recorded there.

Whether those ranges are still appropriate, there may be some reason to doubt. Certainly as at the time they were recorded it

appears that the lung tissue, at least in fixed tissue, the lung concentration of the fixed tissue, exceeded the therapeutic range in fresh lung tissue.

In other words, Mr. Commissioner, I suggest no more than this: there may be some basis in the toxicological evidence to suggest that the terminal events and death of Jennifer Thomas may have been not merely consistent with, perhaps attributable to dixogin toxicity. It has to be said, of course, the pharmacologist didn't rate very highly the likelihood of digoxin toxicity in this case.

Dr. Hastreiter at the preliminary hearing expressed the view that digoxin toxicity was not likely and Dr. Kauffman gave the child a digoxin score of 1 in his scoring for the CDC.

But notwithstanding that, it is my submission to you , sir, in light of the lack of any clear evidence, to support Dr. Rowe's hypothesis as to the cause of death, the symptoms displayed by the child from 12:30 to 3:30 on the morning she died, the toxicological data, to the extent it may be corroborative of the symptomatology and the presence of the other circumstantial evidence

in light of all those things, you would be justified in finding this is a death which gives rise to some suspicion or concern that it may have been caused by digoxin toxicity and go no further than that, and I would not seek to.

The next child is Barbara Gionas, once again a very sick child. She was admitted to the Hospital for Sick Children on January 23rd, 1981 when she was one day old. She had surgery on January 26th and again on February 18th. She died at 1:45 in the morning on March 9th, 1981 in room 418 having suffered an onset of critical symptoms or terminal events an hour earlier.

Once again members of the Trayner nursing team were on duty.

We go first in this case, sir, to the events on the night when Barbara Gionas died. At page 77 of the chart there is Nurse Trayner's nursing note for the period from 7:30 in the evening until midnight. She records that Barbara's apex was irregular at the time and remained irregular throughout the night. It was irregular in that it was much slower (130's) with short pauses. The ECG strip showed "sinus arrest". I should tell you that Dr. Rowe interpreted that note to mean that what she

was seeing was junctional rhythm where there are no T waves visible on the ECG.

- - - - -

The baby was extremely restless all evening, records Mrs. Trayner, and was very hard to settle. At 45 minutes past midnight she was being fed and was increasingly restless. She was sweaty and clammy and the apex started to fall and she became very bradycardic. A 23 was called and Dr. Soulioti came and gave lasix and atropin.

At 1:00, 15 minutes after that first observation, there was no heartbeat at all. CPR was started and a Code 25 was called.

At the top of page 77 there is Dr. Mounstephen's arrest note. He records when he:

" Arrived child intubated, external massage in progress. IV adrenaline ..."

And so on, no result, asystolic:

" ... intracardiac adrenaline done, no result, asystolic.
Repeated drugs again, no electrical activity.
45 minutes into arrest no electrical activity for 45 minutes. Pupils fixed dilated and no output and the efforts stopped. "

The baby was pronounced dead at 1:45 in the morning.

Now Dr. Rowe agreed that that pattern and those symptoms were consistent with digoxin toxicity, but he also pointed out, and this is found at Volume 18, page 3132 and again at pages 3137, 3138, that on earlier occasions Baby Gionas had exhibited similar symptoms. That is to say on previous occasions the chart discloses episodes of bradycardia, vomiting, occasional nodal beats, atrial flutter, 2 to 1 heartblock and so on. Indeed the last two symptoms, the atrial flutter and the AV block, plus on that occasion some tachycardia and vomiting had occurred on March the 7th. Dr. Kobayashi suspecting the possibility that those symptoms were manifestations of dig. toxicity had ordered digoxin held and had a dig. level done. The previous level on the patient had been 1.9. The level that Dr. Kobayashi ordered was reported back as 1.2 nanograms per millilitre. Dr. Rowe's point of course was this, that this baby could exhibit symptoms consistent with digoxin toxicity even when the level showed that the child clearly was not toxic. Therefore the appearance of such symptoms as part of terminal events certainly was no necessary indication of digoxin toxicity, and it was his view that the symptoms were the product of her congestive

heart failure.

So Mr. Commissioner, we have a familiar circumstantial pattern, final onset and presence of team with the sudden deterioration and the manifestation of symptoms that on the face of it are consistent with digoxin intoxication. When we review the medical evidence, all of the physicians agreed that Baby Gionas' death was consistent with her disease condition. The only physician who expressed any suspicion that digoxin might be involved in the death was Dr. Hastreiter and he was far from confident, and this is found in Volume 81, pages 7526 and 7535.

Now Dr. Kauffman like everyone else recognized that the death and the terminal symptoms were also consistent with digoxin toxicity, but he was unable to express an opinion as to digoxin having caused or contributed to death because the toxicological information was not in his judgement adequate to provide a basis for any such opinion. But he scored the death a 2 with respect to possible digoxin involvement. The CDC for reasons that are not exactly clear catagorized the Gionas death as giving rise to special concern re: digoxin intoxication.

THE COMMISSIONER: I think that was one

of the reasons given by Dr. Kauffman.

MR. LAMEK: That he had given it a 2 despite the lack of clear toxicological information, yes, clearly the symptoms impressed him. There is another case that we will come to.

Dr. Mirkin had the same hestitation that Dr. Kauffman had. The toxicological data are found in Exhibit 95E pages 2 to 3 and all the levels were measured in tissue and material obtained at autopsy after exhumation of Baby Gionas' body.

Now the only items to which I refer and I do so Mr. Commissioner fully cognisant of all that had been said about the difficulties of interpretation of digoxin concentrations in exhumed tissues, the only items to which I refer are specimens T83, specimen of liver; specimen T84, specimen of right lung; T85 specimen of left lung. Now, in each of those specimens, the concentrations measured were above the range of therapeutic concentrations as recorded then by Mr. Cimbura, and well into the range of fatally toxic concentrations as they are recorded for corresponding fresh tissue.

Once again I cannot tell you with confidence whether those ranges remained the last word in the literature. I put it no higher than this

in any event, Mr. Commissioner. Certainly I don't suggest that those concentrations in those specimens standing alone would be any reliable evidence of toxicity. I do suggest that the levels taken in the context of the features that I enumerated earlier may, and in my submission do, justify a finding that a measure of suspicion attaches to the death of Barbara Gionas.

I am going to move to another child, Mr. Commissioner, may we adjourn two minutes early.

THE COMMISSIONER: Yes. 2:15 then.

MR. LAMEK: Thank you.

---Luncheon Recess

---On resuming.

THE COMMISSIONER: Yes, Mr. Lamek.

MR. LAMEK: Mr. Commissioner, over the course of the lunch break I have said to you what I take it is already very obvious that it is very certain I will not complete my submissions today. I have also said to you in light of that and in light of the fact that I am not sure that my voice will last until 4:30 I may ask you to end perhaps at the break today with the chance of completing tomorrow rather than go on until 4:30 today and be voiceless tomorrow. Perhaps I can press on until that stage in any event.

THE COMMISSIONER: It is a case whatever is the break or your voice whichever should come first.

MR. LAMEK: Whichever should come first, maybe the break in my voice will.

We have reached the case I think of Kristin Inwood. This is a baby who was three weeks old and she died at 3:00 a.m. on March the 13th, 1981 on Ward 4B, and once again members of the Trayner nursing team were on duty at the time of the death.

Again she was a sick child and there was a general consensus among the medical experts that her death was consistent with her diseased

condition, but there was some feeling on the part of Dr. Hastreiter particularly that her death was not expected to occur just when it did. I don't intend here to go through the clinical picture because in my submission the factors for and against viewing this death as suspicious or something more are almost independent of the clinical history.

Once it is conceded as it generally is that her clinical condition could have caused her death really the only remaining question is, did it.

There are a number of facts which in my submission are important. The first of them is that the autopsy revealed no clear cause of death. I refer you please to the autopsy report in the chart beginning at page 20. It is the final autopsy report and in particular at page 21, the narrative portion. The penultimate paragraph on page 21 reads:

> "Several factors may have contributed to the death of this infant. However, no clear cause is defined. The infant had cardiomegaly secondary to moderate aortic coarctation. Focal myocardial necrosis presumably on a hypoxic or ischemic basis was present and could conceivably act as

"focus of electrical instability. The lung finding of massive amniotic squame aspiration is significant with respect to contributing to the infant's respiratory distress. This appeared to be in a stage of resolution however and is not a likely explanation for the infant's cardiac arrest."

So the pathologists although they looked around and saw several possible contributing factors could find no clear cause of death. We have heard of course that that is not unusual and I accept that. But whereas here no cause of death is revealed at autopsy, that I suggest hardly calms concerns and stills suspicion about what the cause of death of the child may have been, particularly when there are other factors which may point in another rather less pleasant direction.

The second fact concerns Baby Inwood's death itself and her manner of dying. The sudden onset of rhythm abnormalities, bradycardia, proceeding to arrest, to death and that pattern was generally conceded by the physicians to be consistent with digoxin toxicity.

The third fact, a sample of what appears to have been ante mortem serum was found to

contain 491 nanograms per millilitre of digoxin. You will remember, sir, there was much debate and discussion about the providence, history and handling of that specimen, but in my submission it all came down at the end of the day to this. The material appears to have been serum prepared and stored at the Hospital for Sick Children. The sample may have been frozen or heated at different times, or both. But on the evidence that you have heard neither of those experiences on the evidence you have heard can be shown to render unreliable the results of the digoxin assays on the material.

Miss Cronk pointed out to me that the specimen would be a post mortem specimen of serum rather than ante mortem I think I described it as, I think that is probably right.

Third about this specimen, if the 491 level is a true and reliable level, there is general agreement among the pharmacologists that Kristin Inwood received a substantial dose of digoxin some time before her death. As Dr. Mirkin said, I really don't care if you tell me to divide that number by 4 I am only talking about 125 and that doesn't change my view, but if that number is a reliable number even reducing it by some post mortem

multiplier I still have a level that persuades me there was a large dose of digoxin given to this child.

If indeed Kristin Inwood did receive a large dose of digoxin before her death, and in my submission you are entitled to find that she did, the questions then become; first, could the dose have been administered accidentally or in error. Second, did she receive the dose sufficiently in advance of her death that there could have been distribution to tissues in sufficient quantities to have caused or contributed to her death.

As to the possibility of the digoxin dose having been given by error, it has been suggested that an error may have occurred in the administration of what was intended to be lasix, one milligram IV at 2310 to 2315 on the night of her death. Now that suggestion is of course entirely speculative as to whether such an error ever occurred.

Dr. Kauffman when the suggestion was put to him said and his evidence is found at Volume 72, pages 5846 to 5849 and again in Volume 83, pages 8144 - 8155. He said in his view such an error was unlikely, and in any event he said that that error even if it occurred would not in his judgment have delivered sufficient digoxin to explain the serum

6

or tissue levels found in this child.

As to the second question whether the dose of digoxin played a part in Kristin Inwood's death, or whether it was administered so late, so close to death as to have remained entirely in blood and not to have been distributed through tissue where it could have a pharmacological effect, a fourth fact becomes relevant.

- - - -

DP/WB

The fourth fact, in my submission, which is important is this, that fixed and exhumed tissues of this child contained concentrations of digoxin which, in the opinion of Dr. Hastreiter and Dr. Kauffman are corroborative of the view that digoxin toxicity probably caused or contributed to the deaths. Starting with the levels, sir, found in Exhibit 95A, page 7 to 8, those are the fixed tissue levels and in 95E, page 3, the exhumed. The levels recorded in fixed tissue in Exhibit 95A are in heart tissue and they are levels all expressed as digoxin, that is to say, by assay by RIA following HPLC, 230 nanograms per gram in the left ventricle, 79 nanograms per gram in the left atrium, and 300 nanograms per gram in the septum. Concentrations in the left ventricle and septum are in the area of overlap between the therapeutic and slightly toxic concentrations in corresponding fresh tissue. In Exhibit 95E at page 3, a sample of exhumed thigh muscle was assayed and produced a level of 166 nanograms per gram of digoxin and that appears to be within the fatally toxic range of digoxin concentrations as reported by Mr. Cimbura from fresh thigh muscle.

At Volume 76, pages 6744 to 6745,

BB2

Dr. Hastreiter, although clearly cognizant of the problems of levels in exhumed tissues and presumably he would not want to rely on such levels if they were the only data available, considered that the fixed tissue levels could be looked to as corroborative of the serum level. That is to say, as I understand it, the fixed tissue levels which are in themselves high lend credibility to the very high serum level and by virtue of being high suggest that the digoxin had already distributed to the point of elevating tissue levels. Dr. Kauffman's view, which is stated in Volume 72, page 5846 to 5849, was, as I understand it, essentially to the same effect. He regarded it as corroborative information. It was the view of Doctors MacLeod, Kauffman, Hastreiter and Mirkin that if the serum level of 491 were valid, as to which you have my submission, sir, then a very large dose of digoxin was given to Kristin Inwood. I have referred to such evidence as there is as to the likelihood of such a drug being administered in error. Doctors Kauffman, Mirkin and Hastreiter further opined, again, of course, assuming the validity of the serum level, that digoxin might have caused or contributed to the death of Baby Inwood.

BB3

In my submission, there is no reason in the evidence to doubt the validity of the recorded serum concentration of 491 nanograms and, that being so, you are entitled to find, sir, and I respectfully say you should find, that the concentration resulted from the administration of a very large and unprescribed dose of digoxin as to which, first, there is no evidence upon which a finding of accidental or mistaken administration may properly be based and, secondly, in light of the recorded levels of digoxin in fixed and exhumed tissue and the corroborative use that can be made of those orders of levels, according to Doctors Hastreiter and Kauffman, you are entitled to find that those deaths resulted from that overdose. Essentially, the whole question of Kristin Inwood's death revolves, in my submission, around the view that is taken of the serum concentration.

That, sir, brings me to the last child in this group, Charlon Gardner. This baby, too, died in the early hours of the morning at 4:25 a.m. on March the 18th, 1981, Room 418 on Ward 4A, having suffered a sudden onset of terminal symptoms at 3:45 a.m. Members of the Trayner Nursing Team were on duty on 4 and 4A that night.

BB4

Charlon Gardner was extremely ill. Dr. Hastreiter gave her a severity rating of nine. Dr. Nadas regarded her prognosis as poor. She was regarded as being at a high risk of death, the opinion expressed by Dr. Rowe in Volume 22, pages 4077 to 4078 and Dr. Freedom, Volume 29, page 5481. Once again, there was agreement among all the physicians and pharmacologists that the baby's death was consistent with her diseased condition. Equally, there was consensus that her death was consistent with digoxin intoxication. You will remember, sir, that Charlon Gardner fell into that group of children, described by Nurse Scott, who was stable in the early part of the shift. She went for her break and on her return this child still appeared stable. Shortly afterwards, she suddenly got into very serious difficulty that led rapidly to arrest and death. That evidence of Nurse Scott is contained in Volume 118, page 6903.

It appears from Nurse Scott's note from the long night's shift, March 17 to 18, found at page 57 of the chart, until 3:30 in the morning, Baby Gardner's heart rate was regular and was recorded in a fairly narrow range from 162 to 178 beats per minute. She had a somewhat elevated

BB5

temperature, respiration range between 70 and 50 and there were two apneic spells. The respiration range, at one point, went down to 37. There was rapid, shallow breathing - it is not exactly clear what the timing of the apneic episodes were or of the change in the pattern of breathing.

Dr. Kobayashi was called at 2:30 because of the apneic spells. He then reduced the prostaglandic flow rate. At 3:30, Baby Gardner suddenly became bradycardic and the heart rate became very irregular, Nurse Scott notes. At that point, Dr. Kobayashi was called again at 3:30 when the baby suddenly became bradycardic and the heart beat became very irregular.

His note is on page 60 of the chart. He records: 3:30, called to see baby because of bradycardia and irregular heart rate, 102 per minute, ectopic punctual beats. I have trouble reading the next phrase. Went further bradycardic to 95 per minute and then to ventricular fibrillation. CPR for 45 minutes with intracardiac adreline without success. Code was called in fact at 3:25 and the arrest note is found on page 56. There, there is reference to another observation which does not occur in either

BB6

Dr. Kobayashi's note or Nurse Scott's note, the lower part of page 56, the middle paragraph:

> "Previous sinus rhythm progressed via junctional rhythm, AV block to extreme bradycardia, gasping respiration."

As far as I know, that is the only reference in the contemporaneous notes to AV block having occurred with this child.

> "CPR was commenced immediately with external cardiac massage and (something) ventilation..."

Virtually every trick in the resuscitation book was tried. None provoked any response at all from Charlon Gardner, as set out at the top of page 57, and she was pronounced dead after 40 minutes effort at 4:25 in the morning. There are toxicological findings on this child and they are found in Exhibits 95A and 95B. In 95A at pages 8 to 9, there are recorded concentrations found in fixed heart tissue: left ventrical, 141 nanograms per gram of digoxin; left atrium, 43 nanograms per gram of digoxin and septum, 135 nanograms per gram of digoxin. One should add, in respect to those

BB7

heart tissues, those levels or concentrations in the fixed left ventricle and septum are in the overlap area between therapeutic and slightly toxic ranges recorded in corresponding fresh tissue.

In Exhibit 95B, at page 2, there is reported a concentration in fixed lung tissue of 37 nanograms per gram of digoxin and, so far as relates to the ranges set out by Mr. Cimbura, which were apparently then correct but may not be now, at that time the lung concentration of 37 nanograms per gram of digoxin was beyond the therapeutic range found in fresh tissue and into the fatal toxic range of fresh, lung tissue.

Notwithstanding that and, no doubt, being very careful scientists, Dr. Kauffman gave a digoxin score of one for Baby Gardner and Dr. Hastreiter thought that digoxin toxicity was unlikely.

It is my submission to you, sir, however, that, in light of Mr. Cimbura's findings, the possibility that digoxin intoxication played a part in this child's death cannot be totally ruled out. The events of the death and the circumstances surrounding it may add some weight to

BB8

the digoxin findings. I would like to say that, in my submission, only a relatively low level of suspicion attaches to the death of Charlon Gardner.

Mr. Commissioner, it is clear that we are now getting to the point, if indeed we have not already reached or passed it, where the toxicological information on children is so unclear or so ambiguous that the pharmacologists find difficulty in expressing an opinion on it. You have seen that in the cases, particularly of Dr. Kauffman, with several of the children already discussed. That group of children comprise those who had findings, albeit on fixed tissue, which were comparable, at least in numbers, with the ranges of fatal toxic concentrations in fresh tissue and that, in light of the evidence of Mr. Cimbura, he would expect to see concentrations of digoxin reduced after tissue has been fixed.

CC-1
RD/ac

There are other children for whom we do have toxicological data of greater or lesser value. I propose to go now to the rest of those children for whom there are any toxicological data at all. They are in chronological order of their deaths: Laura Woodcock, Amber Dawson, Brian Gage, Francis Volk, Matthew Lutes, John Onofre, Colleen Warner, and Michelle Manojlovich.

Once we have been through those we are then into children for whom we have no digoxin information, other than digoxin levels taken during life, as part of therapeutic monitoring.

First of all, Laura Woodcock. Her death was a mystery, Mr. Commissioner. That her congenital heart disease was not severe is certainly not sufficently severe to account for her death, was the view of Dr. Hastreiter who gave her a severity rating of 2 and Dr. Nadas, who thought her prognosis was good and Dr. Mirkin. Indeed as I understand the evidence of Dr. Rowe and of Dr. Freedom, they were of the same view as also was Dr. deSa.

At page 33 of the chart is the final page of the final autopsy report and final paragraph which is of interest.

" Congenital heart disease was not

" judged to be a significant problem with respect to the sudden death of this infant. There was a small membranous ventricular septal defect and a persistent left superior vena cava with proximal connection to the coronary sinus. Though the pulmonary valve leaflets were thickened, there was no evidence of pulmonary stenosis or sclerosis of the pulmonary outflow tract. A small haematoma on the anterior mitral valve leaflet, superficial cerebral venous congestion, congestion of kidneys, adrenal medulla and spleen and small serous effusions of mesothelial surfaces reflect agonal changes. The exact cause of the sudden, cardiorespiratory arrest is uncertain. No organisms were cultured from the lungs at postmortem, but the child was on intravenous antibiotics. "

Two things from that final paragraph or that penultimate paragraph.

One, the exact cause of the arrest is not established, but, two, the child's congenital heart anomalies were not judged to be a significant problem in the context of her death. She also had a liver problem, as we know.

You all remember very well Dr. Weber's brief appearance here, but again, that was not considered sufficiently severe to explain her death. Indeed, at the time of her death, the death was not only unexpected, but unexplained. It was reported to the coroner. This is one of those cases in which reporting to the referring physician, Dr. Freedom, described the death as sudden and not expected. Whether that description was apt every time he used it, I do not say, but in my submission it was apt and accurate here.

Dr. Rowe suggested that once the autopsy results were known the mystery disappeared and he considered the findings of pneumonia satisfactorily explained the death. His evidence in that regard is found in Volume 18, page 3058.

I say, with respect to the pathologists, do not appear to have shared that view. I have read the penultimate paragraph of the report and there is no suggestion there, in my submission that the

pathologist regarded findings of pneumonia referred to in the top paragraph of page 53 as being the cause of the death or of the arrest or anything else.

In my respectful submission, Laura Woodcock's death has never been explained on clinical grounds, but die she did and that at 9:40 in the morning on June 30, 1980 on ward 4B. She was one year old.

The Trayner nursing team was on duty on ward 4A at the time Baby Woodcock got into her terminal difficulties. The events of her last night are set out at page 50 in the chart, Nurse Bracewell's note. At the top of the page, June 30th, 1980, at 0400 the apex was regular at 90. Blood pressure 90 over pulse. No respiratory difficulties noted, but she was lethargic. Two hours later, 6:00, blood pressure has dropped to 70 over pulse. Her heart rate has gone up to 100 and is irregular and she has vomited twice. She remains lethargic. Seven o'clock that decline appears to be continuing. Blood pressure is now down to 62 over pulse. Heart rate is down to 86 and is irregular and she is very lethargic and she has vomited again a small amount of mucus.

Dr. Schaffer was there. Dr. Schaffer's note is halfway down the page, 30th of June, 7:30.

" Child looking lethargic, vomiting, irregular pulse. Lethargic, pulse 75, nothing for respiration, blood pressure down to 62. Chest clear. Liver down 4 centimetres. "

ECG he says is recording heart block - atrial ventricular dissociation. The ventricular rate is 70 to 75. There is an impression or differential diagnoses he notes at the bottom of the page: toxicity, electrolyte imbalance, bilirubin toxicity.

I go back to Nurse Bracewell's note far up the page. The course is then followed at 5 minute intervals from 8:00 o'clock. Atropine produced a good result. The heart rate and blood pressure both went up. The baby was still very lethargic, and a couple of minutes after 9:00 the cardiac arrest and a Code 25 was called.

That appears on page 51, in Dr. Rowe's note. Time 9:35:

" Infant had cardiac arrest 0903 hours. Resuscitative attempts continuing, but response poor so far. Sequence of events raises possibility of viral

" type infection and despite the absence of heart failure under observation, the conduction system became involved. Electrolytes and blood gases at the time when arrhythmia started were normal so that it seems unlikely acidosis or respiratory arrest was responsible. The baby was covered by gentamicin for the possibility of sepsis. Other investigative studies were underway and the GI Service had seen the baby. There was no indication of impending decay in the condition and plans were in train to transfer the infant this week to the GI Service. The cause of the episode is thus quite uncertain. "

There was general agreement that in the course of her terminal events Baby Woodcock displayed symptoms that were consistent with digoxin toxicity. Dr. Mirkin regarded the symptoms and especially the heart block in AV dissociation as very telling. His evidence at Volume 87, pages 8963 to 4, and Volume 88, 9054 to 64.

Dr. Rowe agreed that the symptoms of digoxin intoxication were present.

Although Dr. Kauffman gave Baby Woodcock a digoxin score of only one, because of the lack of clear toxicological data and the CDC classified the death as being consistent with the special concern with respect to digoxin.

While at the Hospital for Sick Children Laura Woodcock had only antibiotics prescribed for her, ampicillan and gentamicin. She had been on digoxin at the Oshawa General Hospital, but digoxin had been discontinued prior to her transfer to the Hospital for Sick Children. She had been in the Hospital for Sick Children since June 26th and I see no prescribed or recorded dose of digoxin.

Subsequently, Laura Woodcock's body was exhumed and an autopsy was performed on the exhumed body and muscle tissues sample was sent to the Centre for Forensic Sciences for digoxin assay. Traces of digoxin and/or digoxin-like substances were found, the concentration of 4 nanograms per gram.

In other words, Mr. Commissioner, there are essentially no toxicological data for Laura Woodcock. The finding in the exhumed muscle

is essentially meaningless. A judgement as to the cause of Laura Woodcock's death, therefore, turns in my submission on an assessment of the clinical picture as a whole, and a review of the circumstances surrounding her death. All that we know is, first, her death was unexpected and, second, it was when it occurred, and in my submission, still is unexplained in terms of her clinical and diseased conditions.

Third, it was a sudden death. The final episode characterized by symptoms known to be associated with digoxin toxicity.

Fourth, she got into trouble during the long nightshift when members of the Trayner nursing team were on duty on the other side of the ward, and it is my submission that you may conclude in all the circumstances that the death of Laura Woodcock was on in which there has to be grave suspicion that digoxin toxicity played a part. If it did that toxicity had to result from an unprescribed dose of digoxin, because the baby was not on the drug at the Hospital for Sick Children.

THE COMMISSIONER: I'm sorry, that I hadn't appreciated. The baby had had --

MR. LAMEK: At Oshawa.

THE COMMISSIONER: I see.

MR. LAMEK: My other presumably more reliable source of information, Miss Cronk tells me the source of information on which I relied as to the age of this child is quite wrong. Laura Woodcock was three weeks and not one year old.

I come then to Amber Dawson, sir. In addition to the circumstantial considerations arising from patent, Amber Dawson died at 2:40 in the morning, July 28, room 418. The Trayner team, or members of it, being then on duty.

In addition to those considerations there are certain objective medical facts about this case which taken together may, in my submission, arouse suspicion about the death of this child. First, the death, I suggest, was sudden and unexpected. Baby Dawson had come back to the Hospital for Sick Children because she was failing to thrive. Her earlier surgery had apparently been successful. Her cardiac condition was not such as to make her physicians and nurses believe she was at risk of death. There was surprise and puzzlement that Amber Dawson died.

Second, no clear cause of death was ever identified. You will recall, sir, I know,

that initially no one knew what had carried Baby Dawson away.

- - - - -

DD/DM/ko

You will remember the 4A communications book the cause of Amber's death is something of a puzzle. It was hoped the mystery would be cleaned up at autopsy and in my submission it was not. The autopsy report begins at page 59 of the chart and I refer in particular to page 63 where Dr. Cutz performing an autopsy at the behest of the coroner completed the autopsy form and for the cause of death wrote:

"The immediate anatomical cause of death not determined."

He listed two matters which he called "contributing factors", they were later to be translated into underlying causes you will remember in the coroner's report, they were described as contributing factors: "Congenital heart disease, right hemidiaphragm paralysis".

There is no cause, no underlying cause identified in the autopsy report.

The perforated stomach upon which Dr. Bain relied as the trigger of Baby Dawson's arrest was not even mentioned by Dr. Cutz as a contributing factor much less as a cause of any degree. It is described at the top of page 63 but is in no way referred to as being part of the cause of death.

The third factor, which in my submission

DD 2

is important with respect to Baby Dawson is that the events leading to her death had a sudden onset and included symptoms of digoxin toxicity.

Nurse Nelles' note for the long night of July 27th, 28th is found at page 80 of the chart and clearly she gives, in the middle of the page, a description of the child in the early part of the night shift is not the picture of a totally stable child. Respirations appear laboured at times, up to 62 per minute at 2400, at midnight. The apex ranging between 106 and 130 and regular and lethargic behaviour. Lasix was given IV, hardly the indication of a child in an absolutely stable healthy condition, she was sick and there was no question of that. It doesn't sound like a well baby but I suggest there is still very little in the early part of that shift as described by Miss Nelles to prepare one for what suddenly occurred at 1:30. At 1:30 the heart rate which had been ranging between 106 to 180 was noted to be dropping, it dropped to 79 and below. The baby started to gag with some seizure activity and a Code 25 was called. Page 84 of the chart is Dr. Williams' arrest note in which he records, no doubt with a narration of Miss Nelles:

DD 3

"Sudden recent deterioration leading to collapse. Initial condition gasping spontaneously with aspiration extreme bradycardic."

There was no response at all to the resuscitation efforts. No matter what was done, adrenaline, whatever it was, the same note "no response, still asystole", and after 50 minutes of effort after having produced any electrical response at all in the child resuscitation was discontinued on Dr. Izukawa's advice. That is to say in the manner of her dying Amber Dawson also fit a familiar pattern; sudden onset, a dramatic change in cardiac behaviour, progressed swiftly to arrest and could not be revived. That is the case for suspicion as to Amber Dawson. She fits all the patterns and her death is not satisfactorily explained in terms of her clinical status or her disease condition.

The toxicological information about her is hardly helpful, this is found in 95A at page 11. For the left ventricle, although 19 nanograms per gram of something like dig. was recorded, presumably after HPLC on a subsequent RIA no digoxin was identified. For the left atrium by RIA 15 nanograms per gram of of digoxin or digoxin-like substance were recorded.

DD 4

In the septum 19 nanograms of digoxin-like substances but no digoxin at all. In the lungs similarly no digoxin or some digoxin-like substances were recorded. Certainly no case can be made on the basis of that information that digoxin toxicity played any part in Amber Dawson's death.

Baby Dawson's death in my submission remains a mystery and the cause of the death is plainly something about which skilled and reasonable physicians may disagree and indeed have disagreed. The CDC placed this child in Category A on the basis that her death was unexpected and inconsistent with her clinical status consistent with digoxin intoxication.

Dr. Kauffman couldn't opine on the question of digoxin involvement in the death. The lack of clear digoxin data made it impossible for him to do so, and for lack of clear data he gave her a digoxin score of 1.

Dr. Hastreiter who may be considered by some to be rather a hawk on digoxin related deaths in this matter could go no further than to say digoxin overdose was possible and that there was a fair probability of digoxin overdose.

Dr. Mirkin considered that the symptoms exhibited by the child, vomiting, lethargy, anorexia were not attributable to digoxin toxicity of which he

DD 5

saw no evidence.

There is, I suggest, a nagging question about the death of the Dawson baby and it arises mainly from the inability to provide a cause of death from her clinical condition. At the end of the day that nagging question may be enough to justify your concluding that some level of suspicion is aroused here. In my submission and the CDC to the contrary notwithstanding any such suspicion has to be of a relatively low order.

I am reminded, Mr. Commissioner, and the point is a perfectly proper one, it was also part of Dr. Kauffman's evidence that because the tissues of Amber Dawson had been fixed in Klotz solution for an extended period of time, and since she was one of the first children to die, one of the longest of all, the lack of significant digoxin levels in those tissues is in a sense a non-fact, you can neither infer digoxin toxicity from them neither can you rule it out on the basis of the low level, there is no evidence that propels you in either direction.

The next child, sir, is Brian Gage. Brian Gage died at 4 o'clock in the morning of August 27th, 1980 at Room 418, he was a month old. Once again the members of the Trayner team were on duty the

DD 6

night that he died. Surgery had been scheduled but he died before reaching the OR. I am sorry, I said he died August 29th, he died September 25th, forgive me, surgery had been scheduled for September 25th, the very day that he died and he died early in the morning and of course did not reach the OR.

In a blood sample that was drawn at 4 o'clock the afternoon before he died, a digoxin concentration of 3.5 nanograms per millilitre was measured. You will remember he had received a double dose of digoxin by error that day and the incident report referring to that is Exhibit 308. Digoxin was held as a result of that and it was not resumed before he died. None of the pharmacologists considered the 3.5 level to be related to the cause of his death. The toxicological information on Brian Gage is no more clear than it was on the last two pages that I have discussed. In Exhibit 95E on page 4 there is a report on three specimens obtained from this baby's exhumed body and one specimen of what appears to be the ante mortem serum but it says it was drawn prior to August 11th, 1980, which is rather remarkable since the child was not born until August 27th, 1980, so what we make of that ante mortem sample I do not know. It certainly cannot be taken as indicating any serum digoxin

DD 7

concentration on any given day let alone at or close to the day of his death.

In the exhumed material a muscle sample showed only traces of digoxin or digoxin-like substances. Digoxin was identified in samples of contents of large and small intestines, but according to Mr. Cimbura those levels do not appear to indicate any toxicity although of course he treats them as inconclusive either way because of the serious problem of interpreting levels in exhumed tissues.

Brian Gage was a blue baby, he had congested heart failure which had been controlled by digoxin and diuretics until he could get to surgery. Among the non Hospital for Sick Children physicians there was a difference of opinion as to whether the baby's death could be explained by his clinical condition. Dr. Nadas for the CDC considered Gage's prognosis as guarded. Dr. Mirkin thought the death was to be expected, that the child was showing signs of deterioration, that evidence is at Volume 87, pages 8858-8859. Dr. Hastreiter was the voice in opposition. Although he conceded that it was possible that Brian Gage's death resulted from his diseased condition. He thought the prognosis with surgery was reasonably good. He did not think the child to be at immediate

DD 7

concentration on any given day let alone at or close to the day of his death.

In the exhumed material a muscle sample showed only traces of digoxin or digoxin-like substances. Digoxin was identified in samples of contents of large and small intestines, but according to Mr. Cimbura those levels do not appear to indicate any toxicity although of course he treats them as inconclusive either way because of the serious problem of interpreting levels in exhumed tissues.

Brian Gage was a blue baby, he had congested heart failure which had been controlled by digoxin and diuretics until he could get to surgery. Among the non Hospital for Sick Children physicians there was a difference of opinion as to whether the baby's death could be explained by his clinical condition. Dr. Nadas for the CDC considered Gage's prognosis as guarded. Dr. Mirkin thought the death was to be expected, that the child was showing signs of deterioration, that evidence is at Volume 87, pages 8858-8859. Dr. Hastreiter was the voice in opposition. Although he conceded that it was possible that Brian Gage's death resulted from his diseased condition. He thought the prognosis with surgery was reasonably good. He did not think the child to be at immediate

DD 8

risk of death and considered that he should not have died from his disease.

The cardiologists at the Hospital for Sick Children did consider the death to have been caused by the baby's condition, and Dr. Freedom in particular considered hypoxia to be the cause of death. Dr. Kauffman, Dr. Hastreiter and Dr. Mirkin all considered the death to be consistent with digoxin toxicity although to varying degrees. Dr. Mirkin thought there to be some evidence of digoxin toxicity in the child's terminal symptoms. Dr. Hastreiter not only considered the nature of the symptoms to be consistent with digoxin toxicity, but he also viewed the unexpected nature as he saw it, the unexpected nature of the terminal event itself to be further evidence of toxicity.

Dr. Kauffman thought the death and terminal symptoms to be highly consistent with digoxin toxicity, which is why despite utterly inconclusive digoxin data he gave a digoxin score of 2 to Brian Gage. Although he thought the death to be highly consistent with digoxin intoxication he said he could not with any confidence say that digoxin toxicity was the cause of death, and that of course in light of the lack of clear toxicological information.

DD 9

With respect to the nature of the terminal event itself, may I refer to Nurse Scott's notes at page 65 of the chart. She records that the child is in no respiratory distress on September 24-25. Vital signs were stable, he was not cyanosed. He vomitted the 9:30 p.m. feed and he also vomitted later half strength formula and then later D5W, glucose water. Nurse Scott left him to go for her break. When she came back the baby's condition was as it was when she left him, this was her evidence you will remember. Shortly afterwards she sat him up to burp him, put him down again and his heart rate suddenly plummeted to 49 and was weak.

- - - -

EE
DP/cr

CPR was started, Code 25, the arrest note is found on page 64.

"Called in to see re sudden cardio-respiratory arrest. Apparently had been vomiting tonight and had respiratory distress today. During a feed of clear fluid, suddenly arrested."

Down at the bottom:

"Initial response to resuscitation but bradycardia recurred and did not respond to further resuscitation. Pupils fixed and dilated."

4:00 p.m. child pronounced dead.

What we have here, Mr. Commissioner, is a replay of what is fast becoming a classic conundrum. The death in the view of at least one physician was not really expected or adequately explained on the basis of the child's clinical condition. The autopsy report confirms the diagnosis about congenital heart defects, confirms the evidence of congestive heart failure but does not identify a cause of death. Perhaps that may be taken as some justification or corroboration for Dr. Hastreiter's view. There was a sudden precipitous and irreversible

decline in the early hours of the morning in the presence of one nursing team and we have exterminal symptoms which are consistent with digoxin intoxication so much so, in the opinion of Dr. Kauffman that he scores the death as a 2 for digoxin toxicity notwithstanding the lack of any toxicological evidence of any significance.

Then we have totally unhelpful digoxin tests. Which way the inference is going on that is a difficult question that you have to decide. It is my submission, sir, that those elements taken together, sufficient to justify regarding the death as suspicious, have perhaps a somewhat higher level of suspicion than that which I suggested for Amber Dawson.

THE COMMISSIONER: Before you go on, I notice the time - are you leaving Brian Gage now?

MR. LAMEK: Yes, I am leaving Brian Gage, sir.

THE COMMISSIONER: Do you want to --

MR. LAMEK: I would like not to come back after the break but perhaps I could deal with Francis Volk before we take the break. I will not be long with him.

Francis Volk was three months old when

he died. He died in Room 418 at 5:15 in the afternoon of October 23rd when one or more members of the Trayner team were on duty. Francis Volk was an extremely sick child. He spent the whole of his short life in the Hospital, being transferred to the Hospital for Sick Children at the age of one day. He underwent surgery twice and had severe congenital heart defects and congenital lung defects. He suffered from congestive heart failure. He had respiratory distress throughout his life and latterly was suffering from bronchial pneumonia.

At autopsy the death was attributed to a combination of pneumonia and severe congestive heart failure. On the last night of the baby's life Nurse Scott made the observation which is recorded in her note at page 146 of the chart, Volume II of the Volk chart, the long night nursing note is in the middle of the page, perhaps one-third of the way down: Chest sounds clear after suction, air entry reduced on the right side. Perhaps you could compare that to the top of the page, the long day nursing note: chest good air entry after physio otherwise very rattley. Evidently by the time the night time came around there was reduced air entry on the right side. The colour was pale, although he was in 70 per cent oxygen.

Very sweaty, very clammy throughout the night. Restless. Did not settle until after midnight feed. Respiration:30 to 50 sub sternal intercostal indrawing and he had a difficult time breathing apparently. The apex was regular and in the range between 120 and 143.

On October 23rd of the long day at page 147, Nurse Ganassin's note I suggest establishes the same sort of picture as on the previous night. Child vomited approximately 30 ccs two hours after his last feed at 1400. Apex was regular ranging from 147-137-153 before the arrest. Before the arrest it went up to 200. The same respiratory symptoms, intercostal sub sternal indrawing throughout the day; blood pressure stable; quiet all morning; distressed with bath; settled well. Very restless after lunch. Then a brief terse one line "1650 ceased and arrested. Code called".

The physician's note on page 146 reports, I believe his note is complete on page 145, tne pages are bound out of order, Mr. Commissioner. He reports that he was called to see Francis for vomiting and tachycardia, found a pulse of 160, respiratory rate of 60, pale with indrawn respiration. Then follows the arrest note on page 145. Code 23

was called and it says a full code followed, the IV team. Upon arrival he was without heart beat. He was intubated by the anesthetist, had no pulse, bicarb was given and so on. Went into ventricular fibrillation, an attempt was made for conversion of that fibrillation flat line, did not respond, pronounced dead at 1715.

Other than vomiting, which may well be the least specific of all symptoms I would have thought, I suggest Baby Volk did not exhibit any of the known symptoms of digoxin toxicity. No one suggested that his death was anything other than natural caused by his diseased condition.

In my submission there is nothing in the chart or in the evidence that provides any basis for disagreeing with the unanimous experts' view that this death was a natural one and in my respectful submission, sir, you should so find.

I would be grateful if we could break until tomorrow morning.

THE COMMISSIONER: Until 10 o'clock tomorrow morning.

MR. LAMEK: Thank you, sir.

---Whereupon the hearing adjourned at 3:20 p.m. until 10:00 a.m. Tuesday, June 12th, 1984.